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Section for the Study of Disease in Children.

[*November* 22, 1929.]

Enuresis.—D. W. WINNICOTT, M.R.C.P.—(Abstract.)—Two main groups of patients are seen :—

(*A*) In children of the first group there is increased sensitivity of the urinary tract, with increased frequency or urgency—or both—of micturition, the urgency leading to enuresis. This is a manifestation of the anxiety state, which also accounts for the frequently associated polyuria. Clinically, the children are often over-excitable and restless, and probably in some cases exhibitionism is important. The symptoms here are associated with unconscious phantasies, not necessarily dealing with micturition sensations, but properly belonging to masturbation sensations; and the anxiety derives from the sense of guilt which accompanies the unconscious masturbation phantasies in these children.

(*B*) In children of the second group, the enuresis, more usually nocturnal, accompanies phantasies of micturating, a revival of real or phantasied sensations of infancy, characteristically in relation to some person or situation closely associated with such a person. In this group there is typically no increased urinary tract sensitivity, and it is in these cases that many observers have reported anæsthesia. Clinically the two groups overlap.

Children of the first group are brought to the doctor for fidgetiness, over-excitability, ?-chorea, as often as for the urinary symptoms. Those of the second group are usually brought for the enuresis.

In each group there may be associated defæcation disturbance. In the first group this usually takes the form of periodic abdominal colic, increased urgency of defæcation, or diarrhœa. Such symptoms are fairly common, and occur as anxiety symptoms apart from urinary symptoms. In the second group there may be, but much more rarely, incontinence of fæces. It may be possible here to demonstrate anal anæsthesia. This defæcation disturbance always indicates severe emotional disorder.

Enuresis is one of many symptoms which imply in a patient some degree of failure to reach, without damage to the personality, the full genital phase in emotional development. It is so frequent that one must postulate that some degree of this failure is common, a fact already well known.

[*Dr. Winnicott illustrated his paper by Cases described in detail.*]

Discussion.—Dr. E. STOLKIND asked whether the children's dreams had been investigated. Many children with enuresis had significant dreams. Several of his patients related that they dreamt they were micturating, and on waking discovered that they had actually micturated while dreaming. He agreed that almost all children with enuresis were nervous, often with hypersensitive vegetative system, and, in most cases, they had nervous parents. He had had cases of enuresis in adolescents which had responded to treatment of the nervous system.

Dr. WORSTER-DROUGHT said most neurologists agreed with Dr. Winnicott that, in the majority of cases, enuresis was a manifestation of a psychoneurotic disposition. He himself believed it to be a condition of heterogeneous ætiology. The cases had to be investigated on their individual merits. Surgical conditions should first be excluded, and next medical conditions, such as gross disease of the nervous system, infection of the urinary tract and endocrine disorders. There would then remain hypoplasia of the lumbar part of the cord, which appeared to be a very definite condition, in which there was usually absence of ankle-jerks. If all these conditions were excluded the enuresis was a neurosis.

Dr. T. R. HILL said an interesting point in connection with these children was as to how far they could be said to come within the meaning of what Janet called psychasthenic. In psychasthenia there was apparently an inherited psychoneurotic condition due to emotional

abnormality. Some of the cases were explicable on the basis of an inherited exaggeration of crude effect. These children seemed to be born with an excessive amount of emotion, so that their response to normal stimuli was an exaggerated one.

CASES.

Encephalitis Periaxialis Diffusa of Schilder.—C. WORSTER-DROUGHT, M.D.—H. G., a girl, aged 11 years. First seen February 1, 1928, complaining of loss of use of right hand.

Previous History: Measles, chicken-pox, whooping-cough and scarlet fever.

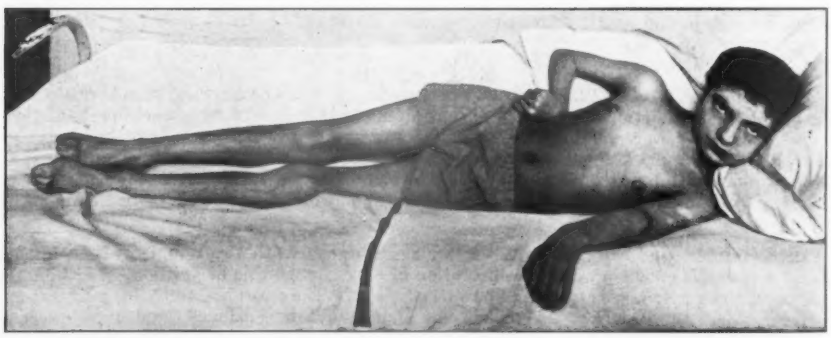
Family History: Nothing of importance. Six other children in family, all healthy.

History of Present Illness: On December 31, 1927, could not properly move the fingers of the right hand. During next three weeks gradually lost use of right arm from below upwards, and right foot was becoming weak.

Condition on Admission; February 1, 1928.—Complete motor paralysis of right arm, with increased deep reflexes; weakness at right ankle; knee and ankle jerks exaggerated; bilateral patellar clonus and ankle clonus; both plantar reflexes extensor.

Subsequently, the following symptoms were noted: irritability, with occasional attacks of hearty and uncontrollable laughter for no apparent reason, and less frequent attacks of crying. Occasional generalized epileptiform attacks. Slow and deliberate speech, sluggish pupillary reactions. Conjugate deviation of eyes to right, with inclination of head to left. Weakness of both external recti, without obvious paralysis; sluggish left corneal reflexes, right-sided facial weakness on voluntary movement. Emotional movements on left side of face less free than on right. Increasing spasticity of right arm, with irregular movements only on attempting to use it. Loss of power and proximal spasticity in left arm, right arm jerks being greater than left. Clonic movements on attempting to use left arm. Right plantar reflex invariably extensor, left variable. Impaired sense of position in right fingers and toes, with diminished vibration sense over right tibia; otherwise sensation has been normal throughout.

Present Condition.—Pupils dilated and equal. Right pupil more sluggish to light and accommodation than left. Primary optic atrophy both fundi. Some



Encephalitis periaxialis diffusa of Schilder. (Dr. Worster-Drought's case.)

weakness left internal rectus. No nystagmus. Hearing normal. Tongue protrudes to left. No sensory loss. The general attitude of limbs is one of muscular hypertonia of the "decerebrate rigidity" type. The right arm is more rigid than the left, and both show hyperpronation of forearms and clonic movements. The arm jerks are brisk, left greater than right. Motor-power in both arms is weak, especially

the right. Lower limbs spastic in full extension ; motor power weak and occasional clonic spasms. Knee jerks exaggerated, left greater than right, bilateral ankle clonus, both plantar reflexes extensor. A sluggish abdominal reflex is sometimes obtained in the upper quadrants but no reaction in the lower. With considerable assistance she stands on her toes with knees fully extended. Sphincters and other systems normal. Mentally, there is impaired emotional control, otherwise no abnormality. Blood : Wassermann reaction negative. Cerebrospinal fluid : normal in all respects.

Commentary.—It is difficult to see what diagnosis other than Schilder's encephalitis is possible in this case. At an earlier stage of the disease the patient was shown at a meeting of the Section of Neurology,[1] with a tentative diagnosis of Schilder's encephalitis. The possibility of hepato-lenticular degeneration, however, was suggested by others. This latter diagnosis may now, I consider, be entirely ruled out. Hepato-lenticular degeneration or Wilson's disease almost invariably begins with tremors of the limbs, whereas the disease in the case shown began with definite paralysis of one hand. Also, in hepato-lenticular degeneration, rhythmical tremors are constant, the muscular rigidity is of the extrapyramidal type, and optic atrophy does not occur. In the present case, in addition to the absence of any evidence of hepatic disorder, there are no rhythmical tremors, the pyramidal system is definitely involved, and there is double optic atrophy. Dr. Kinnier Wilson kindly saw the case and he disowned it as an example of the disease he has described.

In the patient shown, it is evident that the demyelinization has involved the parietal portions of the brain. The disease began in the left hemisphere in the hand area and rapidly spread to the remainder of the Rolandic area of that side. Within a few weeks the right side was also involved, and since that time the symptoms have varied considerably. At one stage there was almost complete immobility of the eyeballs. During July, it was observed that elevation of both arms to above the horizontal invariably occurred when the patient attempted to look up or to speak ; similarly, it was impossible to raise one arm actively or passively without the opposite arm following the movement. Optic atrophy has developed only during the last few months, but as yet the white matter of the occipital, temporal and frontal lobes is not involved.

Discussion.—Dr. E. A. COCKAYNE asked whether the parents were blood relations before marriage. If so, that would strengthen the position taken up by Dr. Worster-Drought.

Dr. W. G. WYLLIE agreed that this was a puzzling case, but he would hesitate to include it in the class of encephalitis periaxialis diffusa, because the cardinal points of that disease were cortical blindness, with no optic atrophy until a late stage, and cortical deafness and mental deterioration coming on with fits and spasticity.

Dr. WORSTER-DROUGHT (in reply) said the parents of the patient were Jewish, but were not blood relations.

He agreed with much of what Dr. Wyllie said, but all depended upon where the process began ; if in the occipital lobe, the condition would begin with cortical blindness ; if it began in the temporal lobe, cortical deafness would be the first symptom ; if in the frontal lobe, there would be mental deterioration, etc. He contended that in this case the process had started in the Rolandic areas, and that neither frontal, occipital, nor temporal lobes were yet affected. Optic atrophy was present, and it was known to occur in many cases of Schilder's disease.

Congenital Absence of Crucial Ligaments, etc. Result after Fascial Graft.—H. A. T. FAIRBANK, D.S.O., M.S. (President).—W. S., boy, aged 9 years.

History.—Seen in 1924 on account of congenital anterior subluxation of right knee. Hyperextension possible to 15°. Some genu valgum. Diminutive fibula

[1] *Proc. Roy. Soc. Med.*, 1928, xxii, 174 (Sect. Neur. 10).

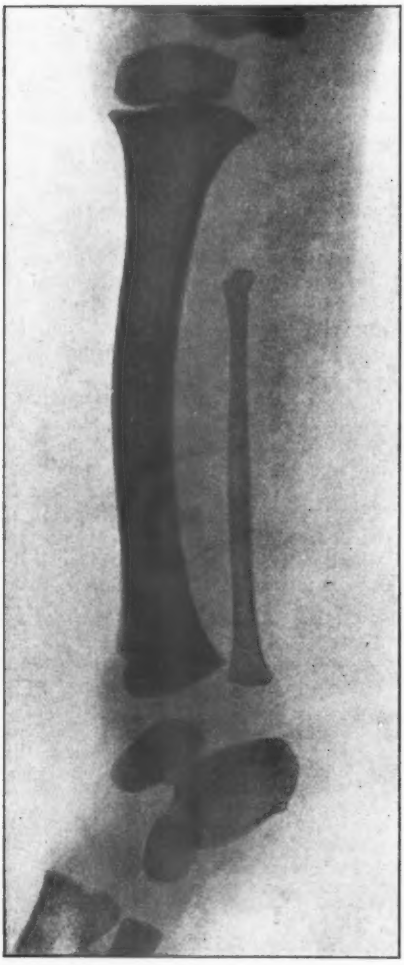


FIG. 1.—Before operation. Note displacement of tibia on femur. (Mr. Fairbank's case.)

(only two-thirds normal width) with marked valgus of foot, dislocation of peroneal tendons, absence of outer part of foot and two toes, and adduction (not abduction) of forefoot. Some antero-posterior curve in lower third of tibia, with dimple over convexity. September, 1924, mid-tarsal and subastragaloid arthrodesis with correction of valgus.

Knee treated by fixation in plaster in flexion for two years, without result.

January, 1927.—Operation on knee. Complete absence of crucials. Anterior crucial made from strip of fascia lata, and internal lateral ligament reinforced by same

FIG. 2.—After operation. (Mr. Fairbank's case.)

(Hey Groves—Alwyn Smith operation). Knee fixed in moderate flexion for four months and then allowed to extend.

Examination.—Present Condition.—Stable knee with no hyperextension and no anterior subluxation of tibia. Slight antero-posterior instability of tibia when knee slightly flexed. Foot stable and in good position. Wears a caliper with a platform for boot to rest on, leg being short.

If the knee remains stable (and it should do so) amputation of the foot, and an artificial leg at a later date, will probably be the best treatment.

Renal Dwarfism.—E. A. COCKAYNE, M.D.—A. D., male, aged 5 years and 5 months. Attended Mr. Eric Lloyd's Out-patient Department, at the Hospital for Sick Children, Great Ormond Street, and was transferred to my care in April, 1929, on account of knock-knees, first noticed in the previous month. Small for age,

Weight 29 lb. in April, 1929, and 32 lb. in October. Epiphyses of long bones thickened. Skiagrams show epiphyses of femora, tibiæ and fibulæ much broadened and with very irregular and imperfect ossification (see fig.). Similar changes in bones of arms but to much less degree. Urine always pale and clear, varying in specific gravity between 1002 and 1009. No albumin found on examinations at first two

Renal dwarfism. (Dr. Cockayne's case.)

visits (acetic acid test) but there has always been a trace or definite cloud ever since. No casts, no pus. In April, blood-urea was 119 mgm., calcium 7·8, phosphorus 4·7, and cholesterol 202 mgm. per cent.; in October, blood-urea was 112, non-protein nitrogen 78·4, calcium 8·4, and phosphorus 5·38 mgm. per cent. Heart not enlarged; no vascular changes.

Discussion.—Dr. J. KINGSTON BARTON said that the interesting matter was to fix a date for the commencement of the renal disease. He believed that in all these cases the patients had renal sclerosis. The test was not the albumin, but the polyuria; also there was this high grade of blood-urea. This child was said to have had diphtheria at 3½ years of age, and diphtheria was often followed by renal disease, usually of the parenchymatous variety. He considered that the condition in the present case was congenital. The blood-urea was still high, and he thought it would always be so. Rickets, in cases of renal dwarfism, usually

came on after the age of four years. Some of the worst cases—i.e., those of severe knock-knee—came under the care of the orthopædic surgeon when the child was 11 or 12 years old. He asked as to the prognosis, especially as Dr. Cockayne said there need not be changes in the heart. Usually children with congenital renal disease did not live beyond puberty, but in Dr. Samuel West's book a man aged 74 was mentioned who had had renal disease of this kind all his life. Post-mortem examinations had shown contracted kidneys, not the tubular disease.

Mr. H. A. T. FAIRBANK (President) asked whether it was usual to find the changes more marked in the lower limbs than in the upper. This child's wrists did not show anything striking, but at the lower end of the femur there was a decidedly fluffy appearance in the skiagram. Recently he had seen a case of late rickets in which the only parts showing changes were the wrists; the other epiphyses were fairly normal.

Dr. E. A. COCKAYNE (in reply) said he could not answer the President's question. He thought the condition was congenital, and that in all these cases the patients had congenitally small kidneys, but did not have the bony changes or the albuminuria or the high blood-urea until later. The high blood-urea was, he considered, the beginning of the end. This child probably would not live more than a year or two.

Splenic Anæmia.—E. A. COCKAYNE, M.D.—(Previous report of case *Proc. Roy. Soc. Med.*, 1929, xxii, 1252.) Splenectomy was performed by Mr. Eric Pearce Gould in July, 1929.

Dr. S. L. Baker's report is as follows: "Enlarged spleen. Sections show a somewhat congested condition of the blood-sinuses, the walls of which are more prominent than normal, the general appearance resembling in some respects that seen in some cases of Banti's disease. The lymphoid tissue is well developed (prominent lymphoid follicles could be seen on the cut surface with the naked eye). There is no evidence of tuberculosis or other specific inflammation. Portions of the spleen ground up and inoculated into a guineapig failed to produce disease."

Since the operation the patient has improved very much in general condition and appears to be no longer anæmic. She has had no further attacks of pyrexia.

Discussion.—Dr. F. PARKES WEBER, speaking in regard to the question whether it was necessary that in every familial hæmolytic case there should be abnormal fragility of red cells, said that in some of the big family series, doctors had taken great trouble to see and examine all the members of the family. In that way many "incomplete" cases had been discovered. He thought that abnormal fragility of red cells was not invariably present.[1] In regard to the microscopical characteristics of the excised spleens, Dr. J. W. McNee (*Glasgow Medical Journal*, 1929, cxi, pp. 65, 193, 288) should be consulted.

Dr. COCKAYNE (in reply) said that amongst the members of a family who had typical hæmolytic jaundice there might possibly be some with an enlarged spleen yet without undue fragility of the red corpuscles in hypotonic saline. Although this fragility was considered by many to be pathognomonic, it was obvious that the red corpuscles could never be in hypotonic saline in the body, and therefore their destruction, during life, must be due to some other cause.

If Dr. Parkes Weber was right and in some members of a family there was no undue fragility of the red corpuscles in hypotonic saline, this child and her father had hæmolytic jaundice, though without jaundice or undue fragility of the red cells *in vitro*. If not, two hereditary diseases had been confused together, in both of which the red cells were easily destructible in the body, but in one of which the red cells were unduly fragile in hypotonic saline, while in the other the red cells were as resistant as normal cells to its action.

Specimens from a Case of Congenital Atresia of the Bile-ducts.—Shown by MICHAEL ALBURY, M.B. (for Dr. DONALD PATERSON).—*History of Case.*—G. W., female infant, aged 5 months. Attended Dr. Paterson's Out-patient Depart-

[1] Gänsslen, Zipperlen and Schüz, in their great work on hæmolytic jaundice and the "hæmolytic constitution," found that abnormal fragility (diminished resistance) of the erythrocytes was absent in 10% of what they called "compensated cases"—quite apart from extremely slight incomplete cases (*Deut. Arch. f. klin. Med.*, Leipzig, 1925, cxlvi, pp. 1-46).

ment, Hospital for Sick Children, Great Ormond Street, on November 2, 1929, and was admitted under Dr. Frew, with the following history: Jaundice had occurred in the second week of life, and persisted, becoming gradually deeper. The urine was dark and stained the napkins. The motions were always white.

Family History.—Healthy parents: four children (one died from broncho-pneumonia). None jaundiced except patient. Pregnancy and labour were normal. At birth the child weighed 7 lb.

On Examination.—Deeply jaundiced, yellowish-green in colour with stained sclerotics. State of nutrition, moderate. Happy and smiling. Weight 10 lb. 14 oz. Abdomen full; liver extended below level of umbilicus, and was smooth and firm. Spleen palpable, 1½ fingers'-breadth below left costal margin. Urine: dark brown; bile-pigments and bile-salts present.

Blood-count: Red cells 3,012,000, hæmoglobin 50%. White cells 12,500 (*Differential:* polymorphonuclears 26%, small lymphocytes 68%, large lymphocytes 2%, large mononuclears 3%, basophiles 1%). No abnormal cells seen.

Wassermann Reaction: Negative. *Van den Bergh's Reaction:* Direct, delayed positive. Indirect, 21 units of bilirubin.

Progress.—November 6, 1929.—Child vomited and passed frequent motions. Next day vomiting occurred again. The weight was now 9 lb. 9 oz. (compared with 10 lb. 14 oz. on admission on November 2).

November 7, 1929.—The child collapsed and died in the night. Slight continuous pyrexia had been present while in hospital.

Autopsy Report.—[Only a partial post-mortem examination was performed].

The body was that of a deeply jaundiced child. *Liver.*—Weight, 220 grm.; greatly enlarged, olive-green in colour, tough, with finely granular surface. Capsule not thickened. On section, the liver was tough and gritty.

The right and left hepatic ducts were traced with difficulty, and were patent as far as their junction to form the common bile-duct. The gall-bladder was represented by a fibro-fatty mass of tissue, from which a fibrous cord, the cystic duct, ran to join the common bile-duct. This also was an obliterated cord of fibrous tissue. The ampulla of Vater was present in the duodenum, but no opening into the common bile-duct could be traced.

Spleen.—Red, firm, enlarged.

Histological Description of Sections.—Liver: Bands of fibro-cellular tissue are well seen, chiefly of mono-lobular distribution.

The typical radial arrangement of liver cell columns is lost here and there, the cells lying in irregular masses about the hepatic ducts, suggesting a reversion to the fœtal pattern.

The new formation of biliary ducts in the interlobular bands is the most striking feature of the section; they are composed for the most part of large cuboidal epithelial cells, without any supporting tissue, growing out into direct continuity with the hepatic cell masses. Inspissated bile occupies the lumina of many of these ductules, some of the bile shows a laminated structure reminiscent of the common pigment stone, in other instances, the appearance suggests a biliary thrombosis of recent origin.

The biliary stasis may be traced back by means of frozen sections to the finest terminal ramifications of the biliary system within the liver cells themselves. For the most part, the Küpffer cells are heavily loaded with pigment. No degenerate liver cells are seen, with the exception of those cells lying at the periphery of the fibrous bands, which show compression and some fatty change.

Spleen.—Shows intense congestion, compatible with portal obstruction. The Malpighian corpuscles are obscured by the large number of red cells.

There is slight thickening of the capsule and trabeculæ.

Remarks.—In this case the developmental defect in the biliary system was fairly extensive, resulting in a complete destructive lesion up to the level of the hepatic ducts. The hypertrophic biliary cirrhosis, associated with congenital atresia, was found.

It is remarkable that this condition should have been compatible with a state of health sufficiently good not to alarm the child's mother, who only sought advice on

account of the deepening jaundice (after five months). Rolleston has called attention to the fact that extreme defect is most often found in patients who have lived for months.[1]

The somewhat rapid death of the child with symptoms of loss of weight, vomiting, coma and collapse, is suggestive of cholæmia, which is a not unusual mode of termination in children who do not die from hæmorrhage or from intercurrent infection.

Discussion.—Dr. MICHAEL ALBURY said that congenital atresia of the bile-ducts was a somewhat uncommon disease; it had been recognized for about forty years and about 200 cases had been described. The pathogenesis of the disease was still uncertain.

In 1892 Thomson had first given prominence to the disease when he published a report of forty-nine cases collected from the literature to which he added a case of his own.

In 1907 Rudolf Beneke, in Germany, contributed to the subject, and extended the series of cases to seventy-four. In 1916 Holmes gave an admirable review of the literature, which then comprised about 120 cases. Although much had been contributed to the subject, little advance in our knowledge seemed to have been made, beyond the views set forth in Holmes' paper.

The disease was commoner in male infants, and hereditary influence was exceptional, but it was a curious fact that there were instances in which several infants in the same family had been affected, these infants all dying early from hæmorrhage. Possibly in such instances the condition might have been confused with congenital acholuric jaundice.

Syphilis was a very rare cause of the disease. Atresia of the bile-ducts might occur with congenital syphilis, when it was associated with perihepatitis, a pericellular cirrhosis, with gummatous obliteration of the ducts. The affection of the neighbouring peritoneum by the syphilitic process was responsible for an erroneous impression that atresia of the bile-ducts could result from a primary fœtal peritonitis.

Two explanations had been put forward to explain its pathogenesis.

(1) That the essential factor in its production was a primary congenital malformation of the ducts. Thomson adhered to this view. He believed that the atresia was primary, and that the resultant biliary stasis resulted in an ascending cholangitis, which led to the development of a biliary cirrhosis. In support of this view was the fact that many instances had been recorded in which the disease was associated with multiple congenital malformations. In two cases, transposition of the viscera was discovered, in others such defects as pyloric stenosis, congenital morbus cordis, pes cavus and mongolism were present. Holmes considered the defect in the bile-ducts was analogous to fœtal anomalies met with in other tubular structures, such as the œsophagus, urethra, rectum and vagina. Beneke suggested that the malformation was due to traction exerted by the rapidly growing fœtal liver on its mesenteric attachments producing an "Abschnurung-prozess," which means a "twisting off," and that this prevented invagination of the common duct by ingrowing duodenal epithelium.

(2) The second explanation was that advanced by Rolleston, who considered that congenital deformity played no part in the production of the disease, which was due to a toxin which, circulating in the mother's blood, reached the fœtal liver, and, in the process of excretion by this organ, set up a cirrhosis and a descending cholangitis, which, in the most severe form, produced complete obliteration of the ducts. Rolleston was persuaded to this view because he had observed how infrequently in the adult, chronic obstruction of the large bile-ducts was accompanied by cirrhosis, he therefore felt that the cirrhosis met with in congenital atresia of the bile-ducts *so constantly* must have another explanation.

Against Rolleston's theory was a body of experimental evidence to show that ligature of the common bile-ducts did set up a cirrhosis, and the information obtained by Mangelsdorf (1882) and Ford (1901), who collected over 200 cases of cirrhosis due to biliary obstruction caused in most of the cases by calculus or carcinoma of the head of the pancreas. Ford concluded that biliary stasis, whether occurring in children or adults was responsible for the same sequence of pathological changes in the liver.

In favour of Rolleston's theory was the fact that a number of cases of congenital biliary cirrhosis had occurred in which no lesion in the extrahepatic biliary system had been present.

1 Allbutt and Rolleston's "System of Medicine," iv, pt. i.

Dr. E. A. COCKAYNE said he had always had a leaning towards Rolleston's theory. Both cirrhosis and familial jaundice attacked more than one member of a family, and the difference seemed to be only a matter of degree. If a patient with grave familial jaundice lived long enough, cirrhosis of the liver might develop, but cirrhosis occurring without stenosis of the ducts was very difficult to explain on a developmental basis. Even if one invoked a maternal toxin, the hepatic system in these children must be abnormally susceptible to the toxin, and that might prove to be the inherited part of the disease. He had seen twins, one normal and the other with stenosis of the bile-ducts. Though this did not settle the question of ætiology, it proved that, if a toxin caused the disease, one child was susceptible and the other was not. Too many familial cases had been recorded for it to be a mere accident. He wished that reports of these cases were accompanied by a statement as to whether the parents were blood relations or not. First-cousin marriages in a population like that of London, were less than one per cent., and if the condition under discussion was inherited as a recessive, one should, in a series of cases, find that, of the marriages that produced them, ten per cent. or more were between first cousins.

Dr. J. H. GIBBENS said he thought it more likely that atresia of the bile-ducts was a congenital malformation. The general impression in this country was that no treatment offered any hope of success, but Dr. W. E. Ladd,[1] of Boston, had recently reported twenty cases, 40% of which were operable, and 30% curable. This was a great advance, and in his opinion surgery should be advocated in all but moribund cases. The complete recovery was against the theory that there was a primary defect of the liver.

Dr. F. PARKES WEBER said he thought the balance of evidence favoured the view that congenital stenosis or atresia of bile-ducts was a developmental malformation analogous to developmental stenosis or closure of the aortic isthmus. In both these groups there was hardly any evidence of familial tendency to the developmental abnormality in question, but there was occasional association with congenital or developmental abnormalities in other parts of the body.

Mr. ERIC A. CROOK said it would be interesting to know whether the thirty-three per cent. of cures applied to this country, as his experience of operative treatment in these cases had been very discouraging.

Dr. ALBURY (in reply) said that he had not inquired for information as to consanguinity. Dr. Cockayne considered that a toxin was at work, but the defect in the present case did not support that view. The extrahepatic ducts were patent and below this level there was complete obliteration of the ducts, the gall-bladder being atrophic. These facts were in favour of a primary congenital malformation.

In reply to Dr. Parkes Weber, the information he used was from Rolleston and McNee's "Diseases of the Liver, Gall-bladder and Bile-duct," (1929) in which the condition was said to be a primary congenital cirrhosis set up by a maternal toxin, with descending obliterative cholangitis. Rolleston thought that in all cases that was the essential change.

The surgery of the condition was first mentioned by Holmes, who said that in sixteen per cent. of cases the hepatic and cystic ducts were patent and normal. Therefore, theoretically, surgery was possible in these cases. It was true that all these children died if they were not operated upon, but, so far, surgery did not seem to be attractive.

Holmes suggested that exploratory laparotomy should be done, and that if anastomosis was possible it should be carried out; if it was not possible they should be drained by means of an external biliary fistula; a repair operation might later be attempted.

In the adult the operation of cholecystduodenostomy presented so much difficulty that it had been mostly abandoned in favour of cholecystgastrostomy. How much more difficult, therefore, must be the technique of such an operation in an infant of a few months old. Seeing that these children were also subject to hæmorrhages, operation became a highly speculative question.

Cyanosis and Finger Clubbing, without Demonstrable Cause.—WILFRID SHELDON, M.D.—Patient, a boy aged 9 years, has been under observation in Dr. Donald Paterson's Out-patient Department at The Hospital for Sick Children, Great Ormond Street, for the last 18 months. Fifth child in family; other children healthy.

[1] *Journ. Amer. Med. Assoc.*, 1928, xci, 1082.

History.—Mother states that clubbing of fingers was first noticed when patient was a baby. Otherwise he was in good health until age of 7 years, when he gradually became cyanosed and short of breath.

On Examination: Weight, 45 lb. (underweight for age). Obviously cyanosed, even veins of conjunctivæ being congested. Well marked clubbing of fingers. Heart not enlarged, and heart sounds normal. Blood-pressure: 108 systolic, 80 diastolic. Nothing abnormal found on general examination. Spleen not palpable.

Special Investigations.—Skiagram of chest confirms normal size of heart; the electro-cardiogram normal. *Blood-count* shows red corpuscles 7,584,000, hæmoglobin 140%, colour-index 0·9, white cells 12,500; differential count normal. Wassermann reaction negative.

The most likely explanation of the symptoms is some congenital abnormality of the heart or main vessels, although there are no signs of cardiac disease. Patency of the interventricular septum is sometimes found after death when physical signs of the condition have not been present during life, and I suggest that some such lesion accounts for the present case.

Cat Asthma. (Treated.)—P. D'ARCY HART, M.B. (introduced by Dr. BERNARD SCHLESINGER).—B. B., a boy, aged 7 years. Seen first at the Royal Northern Hospital, January, 1928. Two years' history of asthma; at any time of day; worse in the summer. No connection with animals noticed by mother or child. No family history of asthma. Skin test: marked positive reaction to cat hair, slight positive to dog hair, negative to all other substances tried.

During the next year had two attacks only; one slight, during measles; the other when playing with a cat. Otherwise avoided cats, the animal which was originally in the house having been destroyed.

Skin test repeated March, 1929: Positive reaction to cat hair, negative to other substances tried.

In April, 1929, a cat was given to the boy to hold: an asthma attack followed.

In August, 1929, he was admitted to the Royal Northern Hospital under Dr. Schlesinger. He was treated by Dr. J. Freeman's "Rush method," being given increasing doses of cat extract subcutaneously at intervals of an hour and a half. After a maximum dose of 85,000 units had been reached—in three days—the boy was discharged.

He has been observed for three months since discharge. During this period he has had no attacks of asthma, although he plays with a cat daily and has been tested with two other cats. The skin test, however, is still positive to cat hair and negative to other substances tried.

Dr. G. W. BRAY said that out of the last 200 cases at Great Ormond Street Children's Hospital, 66 patients said that they had a cat in the home, but in only one-third of those cases was there a reaction to cat hair. It might be that only the presence of a cat brought on an attack: on the other hand a child might be sensitive to egg, cat and feathers: he might have an egg for tea, sit by the fire and stroke the cat, and then sleep on a feather bed and later develop asthma. Absence of one of these factors obviated an attack. In these cases it was very important to ascertain whether any sneezing or lacrymation occurred from the mere presence of the cat, or whether it was necessary there should be actual contact, as in stroking and then rubbing the eyes. In the present case touching was necessary. Another point was as to whether scratches or claw-marks caused an inflammatory reaction, giving a kind of autogenous skin test. The mother in this case said the scratches did not cause a reaction. This was important in prognosis in any de-sensitization methods. In the case of hay fever it was necessary to de-sensitize every year. De-sensitization in the present case, as there was no reaction, could only be a temporary measure.

Acute Collapse of Right Lung.—JOHN GIBBENS, M.R.C.P. (for Dr. J. C. JEWESBURY).—P. R., a girl, aged 7 years. Seen when aged 5 with a history of attacks of cough and dyspnœa every six weeks, the attack lasting about a week. The

first attack was at the age of 1 year. Is always better when in Suffolk. Her grandmother suffers from asthma.

On Examination.—Child looked pale. Respirations 80. Pulse-rate uncountable. Rhonchi heard over whole chest, and breath-sounds over right middle lobe behind were "almost tubular." Adrenalin gave relief. The diagnosis was asthma.

Tuberculin injections were given for a period of seven months during which time the attacks were fewer. In September, 1928, following two bad attacks, adminis-

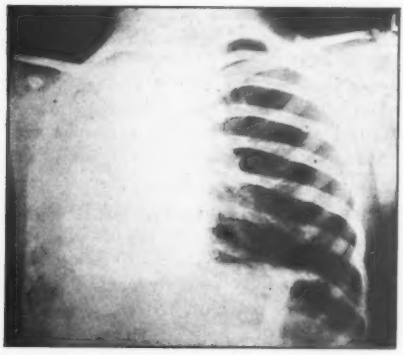


FIG. 1.

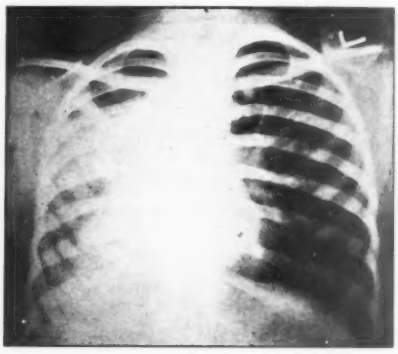


FIG. 2.

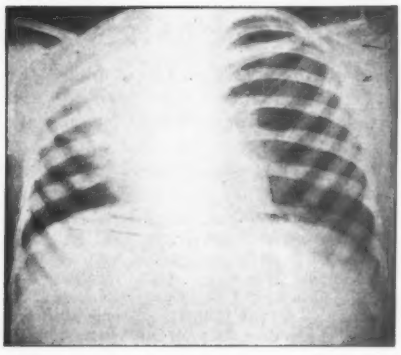


FIG. 3.

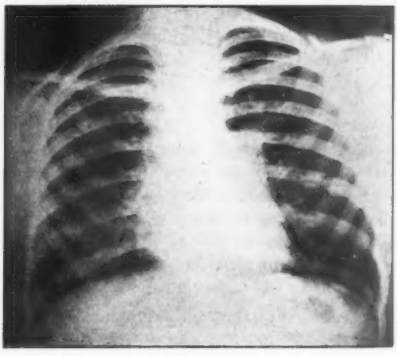


FIG. 4.

Acute collapse of right lung. (Case shown by Dr. Gibbens for Dr. Jewesbury.)

FIG. 1.—Taken 12 noon, October 26. Showing complete collapse of right lung.
FIG. 2.—Taken 11 a.m., October 28. Showing very marked change in two days: collapse clearing up.
FIG. 3.—November 4. Still considerable opacity at right apex.
FIG. 4.—November 20. Right lung completely expanded.

tration of ephedrine was begun and was continued until October, 1929, but without producing much improvement. During the last year the chest has been frequently examined in the intervals but no physical signs of disease were found, although there have been at least nine attacks.

October 26, 1929.—Child brought again with a history of wheezing of two days' duration and abdominal pain, moderately severe, during the previous night. No vomiting. Said to have threadworms.

On examination: Apex beat was in third right space, one inch from right sternal edge. No murmurs. Right side of chest moved better than left, but no dyspnœa or cyanosis. Over right side of chest there was moderate dullness, and almost cavernous breath sounds. No adventitious sounds. Left side of chest showed exaggerated breath sounds. X-ray examination confirmed these findings (see fig. 1). No physical signs of disease in abdomen. Urine, negative; throat clear. The diagnosis was fibrosis of right lung.

Two days later the child came up again. There had been more abdominal pain, also some vomiting, but again the abdomen was completely negative. Apex beat was now in fourth space, under sternum. The dullness over the right lung much less marked, especially high up in axilla and at base. Breath sounds were much diminished, and in some places distinctly tubular. No adventitious sounds. Second X-ray examination made (fig. 2). Admitted to ward, but during next day physical signs remained practically as on admission. No dyspnœa or cyanosis; no more abdominal pain. Two further X-ray examinations while in ward..

Examined again November 4, 1929. Apex beat in fourth space about 1¼ in. from the midline. Percussion note over *both* lungs was now hyper-resonant, breath sounds over right lung were weaker than those over left, but were not tubular; no adventitious sounds. The child looked well and had no symptoms (fig. 3).

A further skiagram, taken on November 20, 1929, shows that the right lung is now completely expanded.

Discussion.—Dr. A. MONCRIEFF said it was easy for a child to have a foreign body in the bronchus without showing any symptoms. The foreign body might take the form of a polyp, or possibly œdema of the bronchial mucous membrane might occur sufficiently to produce this massive collapse.

Dr. J. KINGSTON BARTON said that in the case of a young child, when there was ordinary collapse of the lung, after two years one would have expected to see a greater deformity of the chest.

Dr. K. TALLERMAN said there might be collapse of the lung, with deviation of the heart if there was plugging of the bronchus. In this case there might be a foreign body, or œdema of the bronchi themselves. When the bronchi were clear the lung condition cleared up, and the heart returned to its normal position. A similar thing might occur in pneumonia when there was much exudate. In several cases of that kind the skiagram showed a massive shadow and deviation of the heart towards the affected side.

Dr. H STANNUS said that much of the discussion had been devoted to the question of the collapsed lung, but it was necessary to remember that there were a series of phenomena to be explained, namely, rapid breathing and tachycardia, as well as symptoms which were described as asthma—symptoms that came, went and recurred. Might not such a syndrome conceivably be due to some neuro-vasomotor disturbance akin to asthma (angioneurotic œdema)? A patch of swelling in the bronchus might act, as it had been suggested a plug of mucus might do. He (Dr. Stannus) was led to make these remarks by the fact elicited from the mother, but not mentioned in the notes of the case, that the child was egg-sensitive.

Dr. R. E. SMITH said he might mention the case of a woman who had had attacks of asthma following influenza. She had had every form of treatment, and finally went to Chevalier Jackson's clinic to be bronchoscoped. At the bifurcation of the trachea there were two granulomatous masses, one on each side (she said that sometimes when she lay flat she had an attack of "asthma"). One of the masses was a polyp, which flopped back when she was flat, and this caused collapse of the lung. One of the polyps was removed, and she said that was the first time she felt air entering that lung. He suggested bronchoscoping this patient.

Dr. J. H. GIBBENS, in reply, agreed that a foreign body might cause acute collapse of the lung. Six weeks before admission the child had had some teeth removed, but there was no history suggesting she had aspirated one, and the skiagram was negative. He thought it unlikely that œdema of the bronchial wall could cause collapse: anaphylactic shock, produced experimentally on the guineapig, produced a state resembling asthma, and, post-mortem, acute

œdema of the bronchial wall was found, but here the lung was in a state of acute emphysema, not collapse.

Pneumonia, or broncho-pneumonia with displacement of the heart, was very unlikely in this case, in view of the complete absence of fever, cough, or dyspnœa: the child's respiration rate had never been over 28.

The child was sensitive to white of egg, but attacks had recurred on an egg-free diet. He did not wish to carry out bronchoscopy unless there was another attack. Tuberculin had been used non-specifically without effect; a tuberculin test had not been done.

Postscript.—The child was examined again on December 20, 1929. There were no abnormal physical signs in the lungs. She had been perfectly well for a month.—J. H. G.

Juvenile Myxœdema.—R. E. SMITH, M.B.—P. W., girl, aged 8 years. Youngest of seven healthy children. Parents healthy.

Whooping-cough and chickenpox in infancy. Her mother dates the present trouble from an attack of measles when she was aged 5. Since then she has not

Juvenile Myxœdema. (Dr. R. E. Smith's case.)

grown and her expression has changed so much that she is known locally as "Funny Face." Her hair became brittle and most of it fell out. Tendency to constipation. Grew listless. Intellectually she is considered backward, but is very sensitive.

Condition on Admission.—Clay-coloured complexion, dry scaly skin, height 39 in.; weight 30 lb. Expression typical of subthyroid condition. Temperature (rectal), 97° to 99° F. Pulse averages 80. Skiagram of carpus shows two centres for the os magnus just appearing. Absent on left, corresponding to condition seen in average children of from 4 to 6 years of age. Basal metabolism (estimated by Dr. E. C. Warner) is 35% below normal.

Treatment.—She is now having ½ gr. of thyroid extract (sicca) four times a day. Pulse averages 100; temperature swings from 97°-100° F. She is losing weight, and she is much brighter and more cheerful.

Erythrœdema Polyneuriticum.—R. E. SMITH, M.B.—A. M., a girl, aged 8. Sixth of seven children; four died from pneumonia; two others healthy. Parents healthy. Mother had femoral thrombosis one week after birth of patient.

Previous History.—Bottle-fed on Ambrosia. Bronchitis at age of six months; scarlet fever at age of one year, followed by whooping-cough. In hospital in November, 1923, on account of diarrhœa lasting fourteen days, followed by vomiting. Was unable to speak and made no effort to do so. Could not stand. Temperature ranged between 98° and 102°. A diffuse erythematous rash, most marked on arms and legs, but absent from trunk, was present. Despite her apathy, it was thought she had anæsthesia over the limbs. The rest of the central nervous system was normal. She smelt strongly of mice. Since this time she has had several similar though milder attacks.

Re-admitted to hospital December, 1928, with a temperature of 104°, which gradually fell in four days to normal. Was prostrated, and kept her head continually away from the light. Her "mousy" odour was well marked, and four baths failed to change it. Reflexes were normal, but she was insensitive to pin-prick over lower extremities. She had a sore throat, a swab from which yielded a culture of *Bacillus hoffmanni*. Apparently no vitamin deficiency in her diet. Mentally she was very backward. Erythematous rash was and is still present over the extremities. At no time was there an œdematous condition with the rash. It is suggested that this is a recurrent case of erythrœdema polyneuriticum.

There has been no subsequent attack.

I wish to thank Dr. H. C. Cameron for permission to show these cases.

Two Cases of Convulsions (shown for diagnosis).—J. MINDLINE, M.R.C.S., L.R.C.P. (for Dr. HUGH THURSFIELD).

(I) R. T., aged 6 years, male. Admitted to hospital, October 20, 1929, with a history of numerous fits (five to seven a day) for the past three weeks. Apparently in good health before then. The fits last about half a minute and affect the right side more than the left. The earlier fits were purely tonic, the later ones terminated in clonic spasm. No biting of tongue, incontinence, or cyanosis. Consciousness apparently not lost. No family history of epilepsy or mental disease. Patient was troubled with thread-worms last year.

On Admission.—Healthy-looking child, with marked ataxia in walking, swaying to right; some weakness of right side following a fit; arm- and leg-jerks present and equal; plantar responses flexor; pupils equal and react to light and accommodation; discs healthy. No ear disease. Heart and lungs normal. Urine normal. Cerebrospinal fluid not under increased pressure and normal. Wassermann reaction negative. Skiagram of skull normal. Temperature, pulse and respiration normal. Plantar arches exaggerated.

Treatment.—Sodium luminal ¼ gr., and potassium bromide 10 gr. t.d.s. Fits have gradually ceased; no fit since November 8, 1929. Patient getting up, and ataxia is less marked.

(II) A. J., female, aged 1 year and 3 months, admitted to hospital on October 1, 1929, with convulsions.

Two hours before admission she fell off a chair on to the back of her head. She was not apparently hurt, but two hours later began to vomit and had twitching of face and pyrexia.

On Admission: Temperature 105°, pulse 160, respirations 52. Flaccid palsy of left side, with nystagmus. Shortly afterwards twitching of left side recommenced,

followed by right-sided fits, with nystagmus to right. These persisted for forty-eight hours and were succeeded by paralysis of right limbs and face. Left palsy had cleared up by then. Some blood-stained discharge from nose, but no bruising of scalp. All deep reflexes present and equal. Abdominal reflexes weak on left side, absent on right. Pupils equal and react to light. Discs normal. Cerebrospinal fluid not under increased pressure and normal. Skiagram of skull shows no evidence of fracture or other abnormality. Urine healthy.

Course: Temperature normal after four days, during which time patient remained in semi-comatose condition, but gradually recovered consciousness. October 23, 1929, fresh rise of temperature with vomiting. No further fits. Cerebrospinal fluid still normal. Wassermann reaction negative. Flaccid palsy on right side persists. Reflexes as before. Temperature normal after two days. No otitis. Discs normal. Since then paresis has diminished and face is practically normal. Some spasticity now appearing in affected limbs.

Spasmodic Head Retraction following Encephalitis Lethargica.—T. R. HILL, M.B., and C. WORSTER-DROUGHT, M.D.—E. M., male, aged 15 years.

The onset of epidemic encephalitis occurred insidiously during spring of 1924; beginning with slight fever and attacks of vomiting. The patient then became inattentive at school and had a tendency to fall asleep in class. Concurrently, nocturnal restlessness and noisiness developed.

Spasmodic head-retraction following encephalitis lethargica. (Case of Dr. Hill and Dr. Worster-Drought.)

Since onset of illness patient has exhibited mild behaviour disorder showing restlessness and abnormal aggression towards other boys. Posture is one of slight flexion suggesting Parkinsonism. Intelligence well preserved. In December, 1927, there was a gradual onset of spasmodic movements of head and face. When patient came under observation in 1928, condition was as follows: He suffered from sudden

muscular spasms, repeated frequently, during which head was extended backwards, mouth was opened, and left side of face was contracted as in smiling. Duration of spasms was not more than a few seconds and they have now become slightly milder and less frequent. They can be controlled voluntarily fairly well for a time and are best elicited when the patient walks up and down.

Physical examination shows no other abnormality, except a sluggish pupillary reaction to accommodation.

Commentary.—The diagnosis in this case can lie only between a hysterical manifestation and a chronic encephalitic spasmodic condition. The consideration of the history leaves no doubt that the patient suffered from an attack of epidemic encephalitis. Also, the movements in no way resemble either those of myoclonus epilepsy or lower motor neurone irritation, nor are they atheto-choreic in character. The condition present can be compared with that of persistent tonic head-retraction, which is occasionally seen in chronic encephalitis and may accompany a Parkinsonian syndrome. There is some evidence to indicate that such an extensor hypertonicity is a manifestation of decerebrate rigidity. The case shown suggests intermittent decerebrate rigidity comparable in some measure with the "tonic" fits that may occur in lesions of the mid-brain and pons. This view is further supported by the fact that Wechsler and Brock[1] have recorded a somewhat similar case with intermittent head-retraction, following epidemic encephalitis; during the attacks of head-retraction, however, this patient showed, in addition, tonic extension of the limbs in the exact posture of total decerebrate rigidity.

Scoliosis due to Absence of the Halves of Two Vertebræ.—CARLYLE T. POTTER, M.D. (for Dr. DONALD PATERSON).—Patient, a girl, aged 8, was first noticed to have spinal curvature in October, 1929. Apart from a history of rheumatic pains for the last year, the previous health has been good. Never any complaint of pain in back.

Fourth of family of six. One died from diphtheria; family history otherwise good.

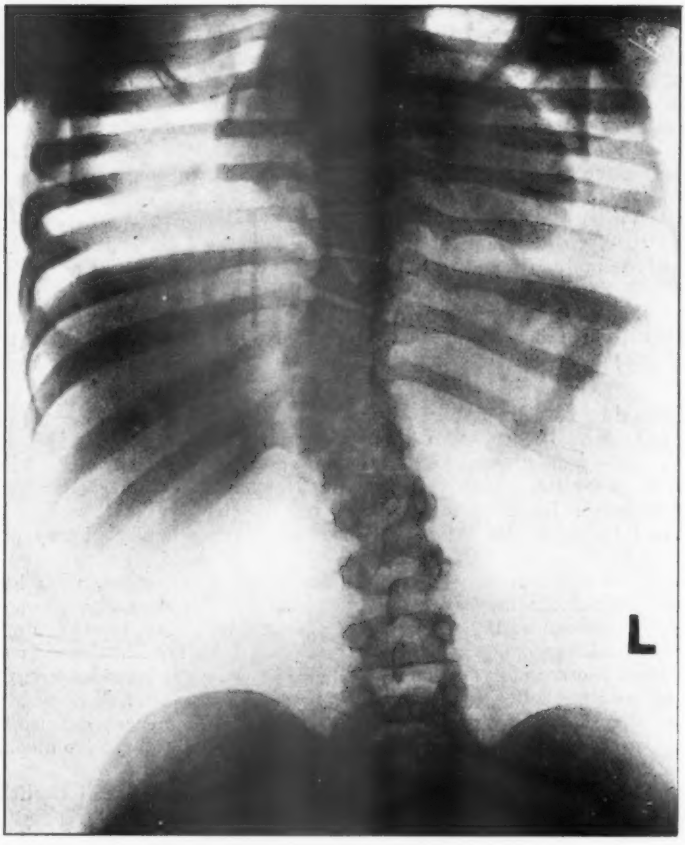
Physical examination shows displacement of trunk to right, with head held usually with slight tilt and rotation to right. Well-marked right dorso-lumbar curvature and pronounced lumbar lordosis. Very little, if any, limitation of antero-posterior or lateral movement of trunk. Transverse plane of shoulders is at angle of about fifteen degrees with transverse plane of pelvis, right shoulder being forward and held higher than left. Right scapula forward and about half an inch further from spine than left. No weakness of muscles of extremities; no sensory disturbance. Left side of chest of slightly smaller girth than right. No displacement of heart or other apparent abnormality.

X-ray examination shows that the left halves of the tenth and twelfth dorsal are absent.

There are only ten ribs on the left side, and the eleventh and twelfth ribs on the right side are fused for about one inch near their articulations. There is deformity of the first lumbar vertebra.

The PRESIDENT said that the only possible treatment would be the use of a plaster bed and a correcting jacket, the idea being to prevent further deformity; he did not think the present condition could be corrected. He could not recommend bone-grafting.

[1] *Arch. Neur. and Psych.*, 1924, ii, 701.



Scoliosis due to absence of halves of two vertebræ. (Case shown by Dr. Potter for Dr. Donald Paterson.

Clinical Section.

[*November* 8, 1929, *continued.*]

Paroxysmal Hæmoglobinuria (?). Case for Diagnosis.—GERALD SLOT, M.D.—E. J., female, aged 34. In childhood had attacks of tetany; was well until a few years ago when her friends noticed attacks of change of colour. Simultaneously her urine has been very dark. During last two years has had severe headache of paroxysmal type and dizziness. Urine has been dark on occasions and contains ? methæmoglobin. At present she is of a greenish-blue tint.

Blood-count.— Red cells 3,700,000, Hb. 67%, C.I. 0·9. White cells 9,400: Differential: polymorphonuclears 68%, eosinophils 2%, large mononuclears, 1%, basophils, 6%, lymphocytes 23%. No abnormal cells. Red cells show punctate basophilia. There is a slight methæmoglobin band in the blood-serum. Van den Bergh (direct) delayed positive; (indirect) negative.

Urine contains a trace of albumin and bile-pigment in form of biliverdin; some urobilin is present. Wassermann reaction completely negative.

The spleen is large and has been so for at least three months. There is no other lymph gland enlargement.

The diagnosis is only tentative. At a period not stated the patient's friends began to notice a change in her colour, which is now an intense greenish-blue, like that of people seen under the ultra-violet lamp. In the intervals between the attacks the spleen is small. The diastase is normal.

There is an interesting link between the faulty calcium metabolism in this case —as illustrated by the low blood-calcium—and the past history of tetany. The suggestion that the patient might be a drug addict is negatived by her doctor, and hospital observation confirms his opinion.

Coronary Thrombosis with Ventricular Aneurysm.—A. J. SCOTT PINCHIN, M.D.—M. G., male, aged 45 (Jewish race). Admitted to Victoria Park Hospital August 17, 1929, from Dr. Burton Wood's Out-patient Department as a case of coronary thrombosis.

History.—Always had good health; served two years in Flanders; no illness while on service, discharged A1. On June 12, 1929, felt severe pain in left chest whilst sitting, radiating over left arm and neck, "felt he would die," very short of breath. Taken to a general hospital; was there four weeks; pain lasted four days. No fits since. Had another slight attack in bed August 7, 1929, lasting two hours.

On Admission.—Sallow, thin man. No dyspnœa when at rest. Pyorrhœa + +. Pulse 80 to 100. Temperature 97° to 99° F. Heart: 3rd rib above, apex beat 6th space in nipple line, ½ in. outside right sternal margin. ? some relative dullness to left of cardiac dullness. Heart sounds very feeble and equal, almost indistinguishable from each other. Blood-pressure 110/92. Arteries in fair condition, dorsalis pedis well felt.

Skiagram (Dr. Sparks).—Shows a bulge of the left ventricle which is expansile and appears to be an aneurysm of the heart (fig. 1, p. 28).

Wassermann reaction: negative (twice). *Blood-count*: Red cells 4,120,000, Hb. 94%. Urine: normal.

FIG. 1.—Coronary thrombosis with ventricular aneurysm (anterior view). (August, 1929.)

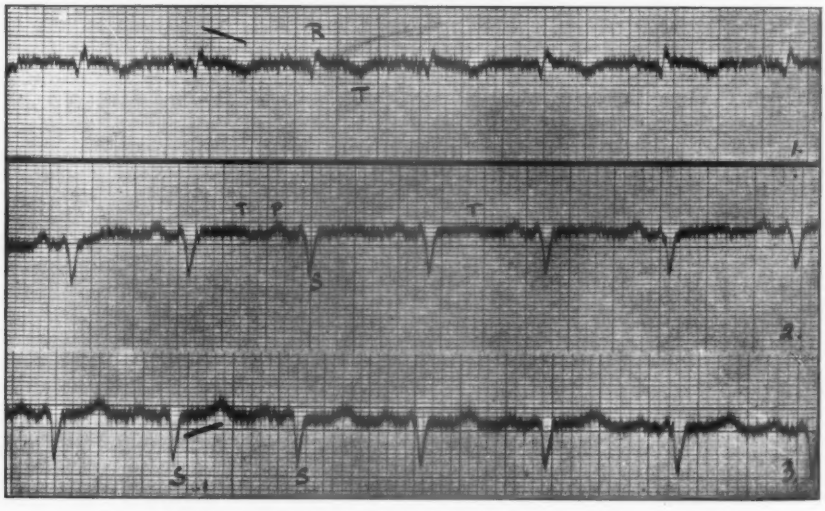


FIG. 2.—Coronary thrombosis with ventricular aneurysm. (September, 1929.)
D^1 shows "high take-off" of R-T, the gradual descent indicated by line \
D^2 shows a flat iso-electric period after S, with T badly marked.
D^3 shows "low take-off" or S-T deviation, the gradual ascent indicated by line /

Cardiogram.—Left side relative preponderance. P2 and P3 large. D.1.: R-T deviation with T negative. D.2.: S-T iso-electric with little indication of T. D.3.: Slight S-T deviation T positive (fig. 2, p. 28).

The findings are consistent with a recovering coronary thrombosis.

If the aneurysm has arisen as the result of the thrombosis, it is of considerable interest in view of the short history (two months).

Variable Heart Block with Cyanosis. ? Specific Pulmonary Arteritis (Ayerza Syndrome) or Congenital Lesion. Case for Diagnosis.—A. J. SCOTT PINCHIN, M.D.—J. H., male, aged 23, admitted to Victoria Park Hospital in 1926.

History.—Father alive and well. Mother dead (neoplasm). Two brothers, two sisters, alive and well. Had a fever, said to be rheumatic, at age of 5. Well till eight years ago when he had influenza. Never well since. Frequent bronchitis, slight hæmoptysis, dyspnœa, cyanosis and precordial pain.

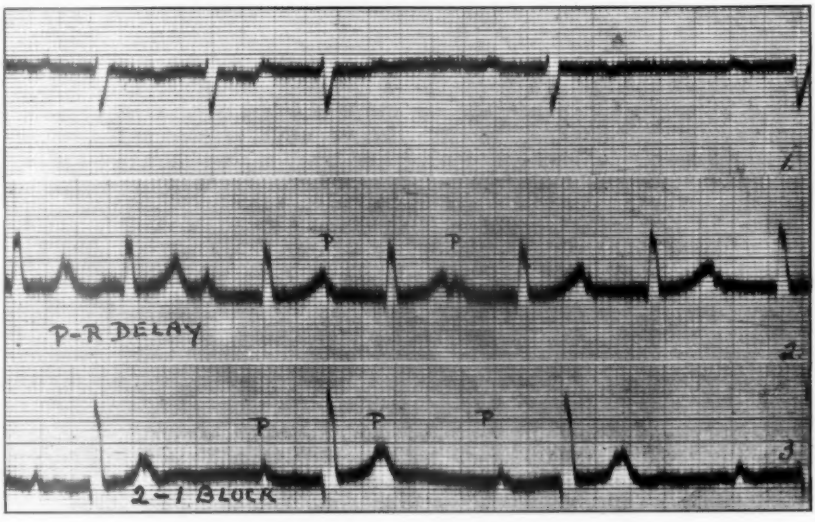


FIG. 1.—Variable heart block with cyanosis. (June, 1928.)

Condition on Admission.—Extremely cyanosed, fingers clubbed. Heart: 3rd rib, apex-beat 5th space 4 in. from mid-line, right sternal margin. A soft, low-pitched systolic murmur over most of precordium. First sound reduplicated at apex; second pulmonary sound greatly accentuated; aortic normal. Pulse 40 to 60. Blood-pressure 102/70. *Blood-count*: Red cells 8,500,000, Hb. 128%. White cells 6,000: Differential: Polymorphonuclears 59%, eosinophils 1·5%, large mononuclears 8·5%, lymphocytes 33% (Dr. Gloyne). Temperature subnormal. Sputum: T.B. negative.

Cardiogram.—Well-marked right side relative preponderance. Heart block varying from delay to complete block. Bundle branch lesion (fig. 1).

Skiagram (Dr. Sparks).—Marked enlargement of heart to left, intermediate zone on left border has a distinct bulge and in oblique view is seen a much dilated atheromatous pulmonary vessel. Hilus shadows extremely dense (fig. 2).

At this time provisional diagnosis of heart block and Ayerza syndrome was made. Since then patient has had various courses of anti-syphilitic remedies, Wassermann reaction now negative. Degree of block varies without apparent cause. General condition somewhat better but last admission to hospital was on account of pleurisy. Heart now 5 in. from mid-line.

A long diastolic murmur is heard after much accentuated pulmonary (?) pressure reflux. Blood-pressure 94/78. Skiagram shows more atheroma in pulmonary vessel (fig. 3).

FIG. 2.—Oblique view. The arrows indicate the greatly enlarged pulmonary artery; over the upper half of the circumference atheroma is well marked. (August 8, 1926.)

The variation in the degree of block, which has been continued over a period of years, is difficult to explain; atropin and barium chloride have been tried without effect, there can scarcely be a syphilitic scar, and if the effect is not vagal—which seems unlikely in view of the failure of atropin—it would appear most likely that it is due in some way to pressure of varying degree on the bundle; I have not seen this occur in the main division of the bundle, but I recorded a case some years ago which had alternating block of right and left branch.

The pulmonary second sound is the loudest I have ever heard, and suggests a high-pressure dilatation in the pulmonary vessel such as is met with on the aortic side in aneurysm; this increase has been described in patent ductus arteriosus.

The skiagram of the heart does not give much evidence of right-side enlargement, though the cardiogram does. In addition, in this case there is a marked scoliosis which may have caused some displacement of the heart to the left. But as a right-side enlargement would be expected in either condition, this does not much affect the diagnosis.

FIG. 3.—Antero-posterior view, showing the marked increase of hilus shadows associated with this condition and the bulge in the intermediate zone due to the pulmonary vessels, the latter indicated by arrows. (October 19, 1929.)

I consider that the marked accentuation of the pulmonary second sound, the absence of thrills or conducted murmurs, the dilatation of the pulmonary vessel and the extensive hilus shadows and changes in the parenchyma of lung, together with the positive Wassermann reaction, point rather to the diagnosis of pulmonary arteritis.

Dr. EVAN BEDFORD said he too thought that the case was one of pulmonary arteritis, most probably syphilitic, and acquired rather than congenital. He had never before seen a calcified pulmonary artery shown in a radiogram, and such a condition must be very rare; it was difficult, even in the aorta, to demonstrate calcification by X-rays.

Hæmophilia Treated by Liver Extract.—TERENCE EAST, M.D.—Male, aged 30. One of four brothers three of whom are sufferers. Has had frequent effusions into various joints, chiefly knees and elbows. Hæmaturia has occurred once. Recently there have been several almost spontaneous subcutaneous

hæmorrhages. Patient began taking cooked liver on September 19, 1929, and liver extract has been given since October 3, 1929. Dates and coagulation times (Dale and Laidlaw's method: Normal, 1 min. 45 sec.): —

September 29, 1929	9 min. 45 sec.	...	October 17, 1929	5 min. 0 sec.
October 10, ,,	6 min. 45 sec.	...	,, 24, ,,	6 min. 40 sec.

Several platelet-counts have shown a considerable reduction in numbers, and this reduction may account for the spontaneous hæmorrhages.

Skiagrams show osteo-arthritic changes in various joints and cystic spaces in the humerus.

This patient seems to be suffering from a combination of purpura and hæmophilia. Only slight improvement followed the administration of liver extract.

Hysterical Wry Neck.—R. RUTHERFORD, L.R.C.P., M.R.C.S.—Patient, male, aged 23, clerk, complains of "twisted" neck. Duration: One year. No history of injury.

Present Condition.—(*a*) General.—Moderately nourished. Nervous type. Whilst being watched he has a habit of holding the chin in his right fingers, as if plucking at it, and whilst he is so doing, the left sterno-mastoid is felt to be completely relaxed.

(*b*) Local.—Right-sided torticollis. There is a hard tumour at the upper part of the left sterno-mastoid, which disappears on increasing the deformity. No enlarged glands palpable. The hard tumour in the upper part of the left sterno-mastoid is a phantom tumour and is due to hypertrophy of the muscle fibres.

Discussion.—Dr. F. PARKES WEBER asked what was the prognosis in this case, and whether the exhibitor was convinced that it was hysterical, or whether he had considered that it might be allied to a motor tic. It would be interesting to hear the subsequent progress of the case.

Mr. RUTHERFORD (in reply) said the patient had been treated by a psychotherapist. After being hypnotized he moved his head freely in all directions, and it was suggested to him that he was cured. When he himself handled his chin, the spastic condition ceased.

Late Rickets.—R. RUTHERFORD, L.R.C.P., M.R.C.S.—L. C., female, aged 16. Complains of injury to right wrist. Five months ago a fall on the hand was followed by slight swelling next day. No pain complained of.

Past History.—Bottle fed. Walked at 2 years. Intellect normal. "Rheumatism" two years ago. "Broken right wrist" six years ago, no deformity following.

Present Condition.—Moderate development. Menstrual periods regular. Appetite good. Easily tired. No urinary symptoms.

There is no arrest of development of the limb-bones as compared with those of a normal person of the same age. The costo-chondral junctions are slightly enlarged and the tenth dorsal spine is unduly prominent; 1½ in. above the right wrist there is a backward angulation of both radius and ulna, causing a slight shortening from external condyle to tip of radial styloid process. A skiagram shows a wide and fluffy epiphysial line at both wrist-joints; the lower ends of both bones show transverse striations very clearly. These epiphyses are palpably enlarged.

The urine contains no albumin.

There is no radiographic evidence of other epiphyses being involved.

(This case and the preceding one are shown by kind permission of Mr. C. P. G. Wakeley, F.R.C.S.)

Mr. CECIL P. G. WAKELEY said this case was interesting because the wrist-joints were the only joints affected. The other epiphyses were normal. The skiagrams showed marked rickety deformity. It was strange that this girl could have marked changes in the lower end of the radius and ulna, and yet nothing in the knees or other joints. She had had two

fractures of the radius and angular deformity had resulted, but she experienced very little disability and could play the piano well. He did not advise any operative procedure in her case.

Partial Albinism. Splenomegaly.—H. V. MORLOCK, M.D.—A girl, aged 12, sent to hospital because an enlarged spleen was found on routine examination by school medical officer. She had had no symptoms of any kind.

Condition on Examination.—Well proportioned child; very pale silky hair, in parts colourless; eyelashes only partially pigmented; iris light blue; nystagmus; some photophobia.

Chest.—Nothing abnormal discovered. Spleen palpably enlarged to 3 in. below costal margin. Liver just palpable; a few shotty glands in both cervical regions and in axillæ.

Central Nervous System normal; urine normal.

Blood-count: Red cells 4,726,000, hæmoglobin 87%, C.I. 0·9. White cells 6,400: Differential: polymorphonuclears, 73·2 %, eosinophils 5·2%, lymphocytes 20·4%, large hyalines 1·2%. *Platelets* 234,000. No abnormal blood-cells seen. A second blood-count showed no appreciable change. Wassermann reaction: negative. Van den Bergh: Delayed reaction. Quantitative 0·6 units. The red corpuscles fragility curve shows no significant alteration from normal.

Overgrowth of Bones of Face. ? Paget's Disease.—C. HAMBLEN THOMAS, F.R.C.S.—Mrs. L. S., aged 44; fifteen years' slowly increasing swelling of the face. Till three years ago spent all her life in India. Twenty years ago birth of only child; since then not very well, but no definite illness. No miscarriages. Fifteen years

Thickening, with areas of rarefaction, of calvarium. Thickening of face bones but lower jaw is not involved.
(Skiagram taken by Mr. Geoffrey Fildes.)

ago teeth of upper jaw were removed after swelling appeared. One year ago was operated on for removal of large swellings on both sides of the nose.

I thought at first that the condition was leontiasis ossium, and I have pared down the bone so as to make the patient more presentable. I am not satisfied with a simple diagnosis of Paget's disease.

X-ray examination shows involvement of the calvarium and face bones with the exception of the lower jaw. The bones of the pelvis—and also the long bones of the right lower limb, in which she complained of pain—are of normal appearance.

Discussion.—Dr. H. STANNUS said this condition was clinically a case of goundou, a disease often associated with other bony changes, chiefly in the upper jaw. It was essential to establish the relationship, if any, between goundou, Paget's disease, and leontiasis ossium. A similar case had been shown a short time previously before another Section of the Society. The condition had been seen in apes and, in one recorded case, in a pony. In tropical countries it was often considered to be due to yaws.

Dr. F. PARKES WEBER said he had noted in the records that in goundou the upper jaw was sometimes affected, as well as the nose, as in the present case, the radiograms from which also showed a great change in the bones of the vault of the skull. It had been claimed that goundou was a late result of yaws, and it had been suggested that a similar result might follow syphilis. He agreed that the present case resembled cases of goundou. As far as he (Dr. Weber) knew, changes in the calvarium in goundou, analogous to those in the present case, had never yet been demonstrated.

Section of the History of Medicine.

[*December* 4, 1929.]

Two Unpublished Autograph Letters of Edward Jenner on the Subject of Vaccination.

Shown by HERBERT R. SPENCER, M.D. (President).

THE letters are in the possession of the Rev. F. W. Joyce, to whose grandfather, the Rev. T. Joyce, of Henley, they were written. Mr. F. W. Joyce has kindly allowed the Society to publish them. Photostat copies of the letters are preserved in the Library of the Royal Society of Medicine.

In the first letter: "Warren's persuasive little tracts" were written by Thomas Alston Warren, "An address from a country minister to his parishioners on the subject of the cow-pox or vaccine inoculation," 1803, 1804 (fourth edition); "Bob" was Jenner's second son Robert Fitzharding Jenner.

In the second letter: "Ld B" was Lord Boringdon; "Catherine" was Mrs. Jenner.

The Medical Boswells.

By Sir WILLIAM HALE-WHITE, K.B.E., M.D.

IN my description of the Withering letters [1] I said that William Withering's diploma (incorrectly called certificate of admission) as Fellow of the Royal Medical Society of Edinburgh, has the signature of Jacobus Boswell, the biographer of Johnson. It has such a signature, but it is not that of the biographer.

There is a dissertation, *De Malo Hysterico* [2], for the degree of M.D., by Jacobus Boswel, dedicated to his paternal uncle, Alexander Boswel of Auchinleck. The biographer was the eldest son of this Alexander. Therefore there were two cousins, each named James Boswell. Comparison of the admitted signature of the biographer with that on the diploma shows a great similarity. Dr. Comrie tells me that "There is no tradition that I know of regarding Johnson's Boswell as having been a member of the Royal Medical Society, but . . . he would only have to take out a medical class to establish himself as a medical student." In 1775 he was, in fact, attending a course of chemical lectures and experiments by Dr. Black [3], who was Professor of Medicine and Chemistry in Edinburgh.

The General list of members of the Royal Medical Society of Edinburgh [4] has these entries of admission: Session XXVI 1762-1763. Feb. 19, James Boswell,[1] March 26, Wm. Withering. Any member, after performing certain functions in the Society, such as reading a dissertation and paying certain dues, could become a Fellow.

1 Unfortunately, the page of the Society's obligation book in which Boswell signed his name has been torn out, so that I have not been able to get a photograph of his signature therein.

Withering's diploma as Fellow is dated 31st March 1764 and has the signature Jacobus Boswell on it. But the biographer left Great Britain in 1762 and did not return from abroad till early in 1766, and was in Edinburgh in the summer of 1766. He might have been proposed as a member of the Medical Society before he left Edinburgh at the end of 1762, have been elected in February 1763 and have signed the diploma in 1766. But there is no evidence for it. The identity of the Boswell signature as that of the biographer is made further improbable by the fact that in the list of Members of the Society, 1737-1835 there is only one James Boswell.[1]

In the list and in Withering's Diploma the name is spelt Boswell. In *De Malo Hysterico* it is always with one l. Many signatures of the biographer have come down to us but only one with one l, namely that in 1758 [5], but he tells us that his father, alone of the family, prefers one l [6], and the author of *De Malo Hysterico*, though usually using two l's, may have thought to please the biographer's father by using one l when he dedicated his dissertation to him. Johnson almost invariably used one l, but the biographer preferring two l's corrected Johnson's letters.[2]

The biographer wrote much, yet there is no evidence that he was particularly interested in medicine save, oddly enough, in the subject of Hypochondria. The biographer's seventy essays called *The Hypochondriack* [7] show that he suffered from depression but, although he read books [8] about it, he does not show any trained medical knowledge. His attendance at asylums and executions was mere morbid curiosity. He says: "I am no physician" [9], and erroneously states that the pulse is higher in sleep than in wakefulness [10]. We find him reading midwifery but only because he was engaged on a legal case concerning the duration of pregnancy [11]. We cannot therefore consider it probable that he belonged to the Medical Society. Nor does it count for much that among his many friends some were doctors and that he was a steward of the Humane Society [12].

It thus seems clear that the James Boswell who signed Withering's diploma was the author of *De Malo Hysterico* and not the biographer.

Nowhere in the biographer's writings[3] have I come across any reference to his cousin, Dr. James Boswell, of whom hardly anything is known. Dr. Alexander Boswell, of East Sheen, tells me that, according to his Boswell Family Bible, Dr. James was born 28 June, 1744, and died 30 November, 1767; tradition says of septicæmia due to wounding his finger when making an autopsy. Mr. A. B. Roxburgh has very kindly let me see his genealogical tree of the Boswell family, in which the date of Dr. James Boswell's death is also given as 1767. Mr. Roxburgh tells me that in the tree of the Boswell family, compiled by Jasper John Boswell, of Norwich, and published in Leeds in 1900, the eldest son of Dr. John Boswell, brother of Alexander of Auchinleck, is "James Boswell, M.D., not married, died 1787," obviously an error for 1767.

This entry occurs in Temple's Diary [13], Monday, 9th June (1783): "Rode to dinner to Blackadder, Mr. Boswell's." Commenting on this the Editor of the Diary, Lewis Bettany, says: "The country house at Blackadder, where Temple and Boswell dined together, belonged not to the latter but to a namesake of his, physician to the Nabob of Arcot, who had bought the property for £30,000. So Temple says in the letter to Mrs. Temple which he wrote in June or July, 1783." Blackadder is three or four miles north of the Tweed, not far from Berwick, and near Churnside. It might naturally be thought that this namesake was Dr. James

1 In the session 1778-79 John Bosville joined—Bosville was one way of spelling the name of the Boswell family. Withering's son joined in 1796-97.

2 See Boswell's *Life of Johnson*, edited by G. B. Hill, vol. 5, p. 124. Mr. L. F. Powell also kindly pointed this out to me. There are forty ways of spelling the name: see *History and Genealogical Tables of the Boswells*, by J. J. Boswell, vol. I, Preface, privately printed at Leeds, 1904. Kindly lent me by Mr. Roxburgh.

3 The physician, Dr. James Boswell's name does not occur in *Johnsonian Gleanings*, by A. L. Reade, privately printed, London, 1923.

Boswell, but that is not so, for he died in 1767, and reference to Mr. Roxburgh's tree shows that about this time there was an Alexander Boswell, M.D., of Blackadder, who died unmarried; he was one of The Boswells of Balmuto, and of him J. J. Boswell (op. cit.) says: "In India he had made himself a name and position, and returning to Scotland with a considerable fortune, he purchased large estates. . . . He died a bachelor in 1812." There can be little doubt but that the Alexander in the family tree, the Alexander of J. J. Boswell and the namesake who had been physician to the Nabob of Arcot, were the same man.

John Boswell took his M.D. degree at Leiden in 1736 with a thesis on Amber, became a Fellow of the Royal College of Physicians of Edinburgh, August 2nd, 1748, was Treasurer from December 1st, 1748, to December 2nd, 1756, and from August 1st, 1758, to December 1st, 1763. He was elected President December 5th, 1770. No other Boswell has ever been a Fellow. His nephew, the biographer, was fond of him. In 1763 he says [14] "Mr. Johnson, Dempster and Doctor Boswell supped with me. . . . The honest Doctor was chearful and conversable and highly entertained." This Dr. Boswell cannot have been Dr. James Boswell, for he did not obtain the title of doctor till 1766. In another letter to Temple [15] Boswell says "The honest doctor thinks you a good quiet philosopher, a kind of Parson Adams," and he again refers to him [16], "For the sake of the health of my wife and children I have taken the little country house at which you visited my uncle Dr. Boswell who, having lost his wife, is gone to live with his son." In 1780 Boswell alludes to the death of his uncle John, thus [17]: "It pleases me that you express concern for the death of my poor uncle Dr. Boswell. He was a very good scholar, knew a great many things, had an elegant taste, and was very affectionate. But he had no conduct. His money was all gone; and do you know, he was not confined to one woman? He had a strange kind of religion. But, I flatter myself, he will be e'er long, if he is not already, in Heaven." Johnson in November, 1773, "spent one forenoon at my uncle Dr. Boswells, who showed him his curious museum; and, as he was an elegant scholar and a physician bred in the school of Boerhaave, Dr. Johnson was pleased with his company" [18]. Fitzgerald informs us that Dr. John Boswell was a warm Patron of the poet Allan Ramsay, who addressed to him some verses which Fitzgerald prints [19]. J. J. Boswell tells us that he kept a diary (op. cit. p. 10).

Fitzgerald says of the biographer's brother John [20] that he was "apparently a physician," but in 1765 his mother wrote to him as Lieutenant. The biographer, the eldest son, was born in 1740, his brother John came next, consequently if he ever followed medicine he soon gave it up for the Army. There is no real evidence of his having studied it; the suggestion that he did apparently arose from the fact that Boswell, writing to Temple in 1772, says his brother John is gone to live at Newcastle with Dr. Wilson, a Physician there [21].

That some of the Boswell family were friends of the Witherings is shown by the fact that Dr. James Boswell and William Withering joined the Medical Society at about the same time, and that James signed Withering's diploma as Fellow. The friendship between the two families continued into the next generation, for, among the Withering letters in the possession of Dr. K. Douglas Wilkinson, of Birmingham —which, through his kindness, I have seen—is one from W. Boswell dated Edinburgh, 3rd December, 1799, sympathizing with William Withering Junior on the death of his father, and there are other letters from Boswells to him in 1800, 1801 and 1802.

Dr. William Withering was on good terms with the Hectors, some of whom were friends of Johnson's, and with one of whom Johnson stayed, so Withering may have met Johnson.

I have restricted myself to those Boswells in the medical profession at the time of the biographer. There have been several medical Boswells since.

REFERENCES.

[1] The Withering Letters in the possession of the Royal Society of Medicine, *Proc. Roy. Soc. Med.*, 1929, xxii, 1087. [2] *Dissertatio Medica Inauguralis de Malo Hysterico*, Jacobus Boswell, 1766. Roy. Soc. Med. Library, Tracts, B. 117. There is also a copy in the Library of the Medical Society of London which is marked as given to Dr. Simms by his friend the author. [3] *Letters of James Boswell*, collected and edited by C. B. Tinker, Sterling Professor of English Literature in Yale University, 2 vols., Oxford, 1924. Vol. i, p. 235. Letter 147 to Temple. [4] There is one in the Library of the Royal College of Physicians, London. [5] TINKER, op. cit., i, 4, Letter 1, to Temple. [6] TINKER, op. cit., i, 17, Letter 8, to Dalrymple. [7] *The Hypochondriack*, edited by Margery Bailey, Ph.D., Assistant Professor, Department of English, Stanford University, Stanford University Press, California, 1928. [8] *The Hypochrondriack*, op. cit., i, 29, and Essay LXVII. [9] TINKER, op. cit., i, 50, Letter 25, to Isabella de Zuylen. [10] *The Hypochondriack*, op. cit., ii, 111. [11] TINKER, op. cit., i, 239, Letter 148, to Temple. [12] TINKER, op. cit., ii, 392, Letter 273, to Bennet Langton. [13] *Diaries of William Johnston Temple*, 1780-1796. Edited, with a memoir, by Lewis Bettany, Oxford, 1929. [14] TINKER, op. cit., i, 27, Letter 13, to Temple. [15] TINKER, op. cit., i, 120, Letter 63, to Temple. [16] TINKER, op. cit., i, 261, Letter 173, to Samuel Johnson. [17] TINKER, op. cit., ii, 309, Letter 204, to Temple. [18] *Boswell's Life of Johnson*, edited by G. Birkbeck Hill, vol. 5 (Tour to the Hebrides), p. 394, Oxford, 1887. [19] PERCY FITZGERALD, *Life of James Boswell*, vol. I, p. 255, London, 1891. [20] PERCY FITZGERALD, op. cit., i, 255. [21] TINKER, op. cit., i, 187, Letter 112, to Temple, and note.

Section of Ophthalmology.

[*November* 8, 1929.]

Hereditary Ptosis with Epicanthus: A Case with Pedigree extending over Four Generations.

By J. Hamilton McIlroy, M.B., Ch.B., D.Sc.

This case is of interest in that the condition of heredity illustrated is of somewhat rare occurrence, no cases, so far as I am aware, having been published since the comprehensive paper by Usher in the "Annals of Eugenics," Vol. I, Parts 1 and 2, 1926.

The girl, K. F., aged 13, was seen by me at a London County Council clinic, and later at the Royal Westminster Ophthalmic Hospital. She was accompanied by her aunt who herself shows a well-marked degree of epicanthus and ptosis. The aunt is an intelligent woman and it is to her that I am indebted for the details of the family history.

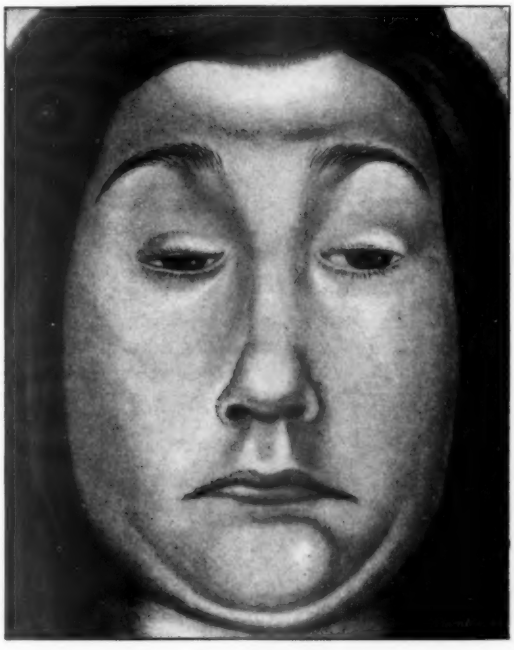


Fig. 1.

I have been unable to examine any other members of the family personally. The girl's mother is said to be mentally defective, and incapable of taking her about; the father was ill and unable to come to the hospital.

There is a well-marked condition of bilateral ptosis with slight epicanthus: the pupils are more than half covered by the lids, and the head is thrown back in the attitude characteristic of double ptosis. There is absence or poor development of the levator muscles of the lids, the eyes being kept open by the action of the occipito-

frontalis muscle (fig. 1). On examination of the profile, one notes a distinct concavity over the supra-orbital region, due to maldevelopment of the bone. The normal furrows of the forehead are wanting.

Nystagmus and divergent strabismus—not uncommon features in these cases—are present.

The refractive condition was difficult to estimate—objectively, on account of the local condition—and subjectively, because the mentality is subnormal if not actually defective. There was myopia of considerable degree, and glasses were prescribed. Right eye: sph. − 5 D. with cyl. − 1·5 axis 30°. Left eye: sph. − 6 D. with cyl. − 1·5 axis 150°, the best vision obtained was $\frac{6}{36}$. She was sent to a myope school in 1922 and has remained there ever since.

Progress in school has been unsatisfactory, but the girl has not at any time been deemed suitable for a class for mentally defectives. She appears to be well nourished. She is emotional and rather nervous, but is said never to have suffered from fits.

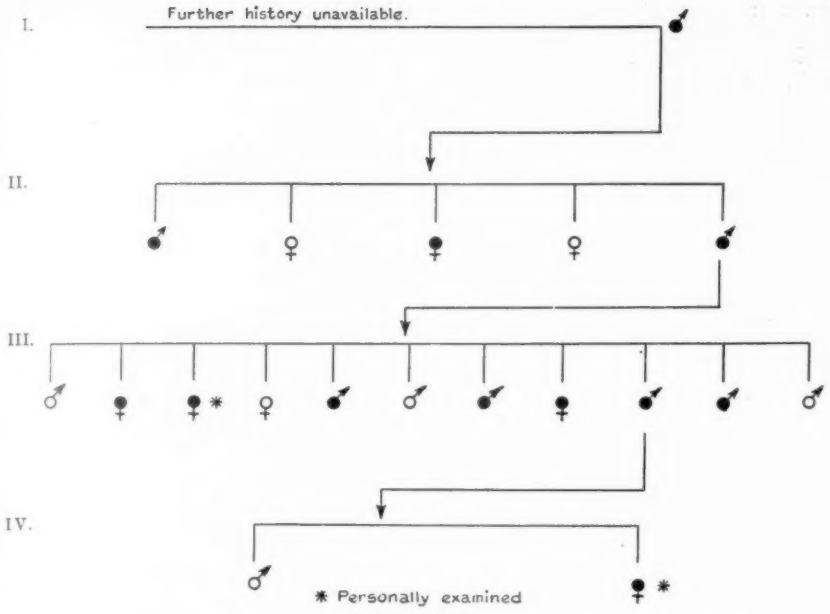
FIG. 2.

The condition of ptosis in the aunt (fig. 2) is rather less pronounced than in the niece. This difference in the aunt is attributed to an operation which was performed when she was 9 days old. She was the only member of the family to receive treatment, her father apparently not having regarded the result as sufficiently successful to make him seek operative interference for the others affected.

(The pupil is not habitually covered by the lids.)

The girl, K. F. (present generation) is one of a family of two; her brother is normal. Her father and his sister (Mrs. M. A. C., whose picture is shown) are of the third generation, of which there are eleven members—four males and three females, affected; three males and one female normal.

It is interesting to note that no member of this generation other than the father of K. F. has transmitted the condition.



Pedigree Chart

(The Chart shows the affected members thus: Males ♂, Females ♀; and the normal, or unaffected, thus: Males ♂, Females ♀).

The previous or second generation consisted of five members (two males and one female affected; two females normal).

The first generation cannot be fully traced; descent comes through an affected male. It is believed that other members of this or previous generations were affected, but the family belonged to Oxfordshire and became scattered, so that precise history is not available.

The points of interest in the pedigree may be summarized as follows:—

(1) There is a definite history of the existence of the condition throughout four successive generations.

(2) The condition has been transmitted solely through the male. Several females were affected but did not transmit further.

(3) No evidence of consanguinity can be traced in any of the parents.

(4) Personal examination was possible in the case of two members only, but it may be assumed that the condition throughout was much the same as that exhibited by these members.

(5) The pedigree comprises four successive generations, twelve members being affected (seven males, five females), and seven members unaffected (four males, three females).

(6) There is an element of mental deficiency in the girl. Her aunt is normal in intellect, but has had numerous fits in recent years, as a result of severe shock.

(7) The colour of the irides in both cases examined is brown. The girl's hair is reddish brown; the aunt's hair is now grey but is said to have been dark. (This bears out Mr. Usher's findings, that brown pigmentation is the rule in these cases.)

Mr. Usher in 1926 summarized the literature up to date, giving a list of authors and pedigree charts for comparison with his own. He personally examined 14 out of the affected 19 members recorded in his pedigree chart and made minute

measurements of the space between the epicanthal folds and of the width of the palpebral fissure.

His chart embraces six generations. Transmission of the condition was by the male, and was mainly continuous, there being a missed generation in two cases.

In practically all the cases there was double ptosis and epicanthus. In the cases of a missed generation only there was an atypical condition, the ptosis being either wanting or one-sided. In all the cases of direct descent (with one exception) the condition was typical.

No consanguinity was noted. The prevailing colour of the iris was brown, and the hair of the head also was usually brown.

This condition of ptosis and epicanthus differs from other hereditary conditions (night-blindness, dislocation of the lens, cataract, etc.), in that transmission is unfailingly through the male.

Mr. Usher touches upon the possibility of two factors for the condition, one for ptosis and one for epicanthus. This would explain the pedigrees of ptosis without epicanthus which have been published, but in all the literature Mr. Usher found only one case of epicanthus unassociated with ptosis.

Epithelial Hyperplasia of the Cornea (? Epithelioma) following Irritation of the Eye by Fuel Dust.—J. W. TUDOR THOMAS, M.D., F.R.C.S.

The patient is a fuel worker aged 52. There is a history of fuel dust entering the right eye three-and-a-half years ago, followed immediately by inflammation of the eye which has not cleared up. I am informed that the fuel dust contained 6% of pitch.

First seen by me August, 1927. There were three separate greyish-white patches on the eye; one on the cornea, one on the inner limbus, and one on the bulbar conjunctiva.

They were slightly raised and had the appearance of greyish-white keratinized material; there was some conjunctival injection. The patient said that these white patches had been present for several months. Two months later the conjunctival patch had disappeared, and the two other patches had merged into one large patch, which extended from the inner side of the cornea horizontally outwards below the pupil, but did not reach the lower margin of the cornea.

In August, 1929, further extension had taken place across the limbus from 4 to 6 o'clock, forming a somewhat triangular-shaped growth about seven millimetres in extent.

Operation (September, 1929).—Growth dissected away from the corneal stroma and removed; a conjunctival flap was drawn over the raw area. Three weeks later a white patch began to form at the limbus at 7 o'clock, and in the course of a further three weeks this had extended along the edge of the conjunctival flap to 3 o'clock on the corneal margin. The white marginal growth was about 2 mm. broad. Vision in this eye was $\frac{6}{36}$.

Microscopically, the growth showed great thickening of the epithelium with a thick stratum corneum, and papilliform downgrowths of epithelial cells with leucocytic infiltration at the base. Two cell nests can be seen in the section, and prickle cells are numerous.

The condition does not appear to be one of xerosis; the patches were not frothy in appearance and were not easily removable.

There was no condition of exposure of the eye such as would lead to the formation of epidermoid cornea, although there was primarily a definitely irritative condition. It is well known that fuel dust and pitch-containing materials cause much conjunctival irritation. Comparing the appearances in this case with those described in congenital epidermoid plaques of the cornea, one notes in the former, both microscopically and clinically, a tendency to assume malignant characteristics that is absent in the latter.

The original appearance of three separate areas, of which one disappeared and the other two fused, does not quite correspond to what one expects to find in a malignant epithelioma.

It seems probable that the original injury with fuel dust was an ætiological factor in the production of the growth. If this is so, then one is tempted to compare the corneal condition in the case described with those changes in the skin that are caused by pitch irritation. One of the changes so produced is a pitch wart.

Epithelial Hyperplasia of Cornea. For description of Section see text.

Pitch warts may be multiple, and are known to have a tendency to become mildly malignant, showing microscopically down-growths of epithelial plugs, numerous cell nests, increase of the stratum corneum, and basal leucocytic infiltration.

Bearing in mind the slight tendency that there is for corneal tumours to penetrate deeply, the microscopical appearances in this case show some similarities to those found in pitch warts. A pitch wart has mild malignant potentialities and the recurrence in this case shows a similar tendency.

It is interesting to note that this patient had a "pitch wart" on his face sixteen years ago. This was destroyed by caustics.

Mr. TREACHER COLLINS said he thought the growth was an epithelioma of the cornea. It was like the epitheliomata in that position which he had previously seen. A characteristic of growths so situated was that they did not tend to dip down into the deep tissue as readily as did epitheliomata of the skin, because of the dense character of the fibrous tissue beneath. The clinical appearance in the case, the recurrence after removal and the histological characters were all in favour of the growths being an epithelioma. His advice was that it should be shaved off as closely as possible to the substantia propria, and then treated with radium. Some years ago he recorded a case which had been treated in that way; only one application of radium had been given. He followed that case up for many years, and when the patient ultimately died there had been no recurrence.

Complete Atrophy of Retina and Choriocapillaris.—LESLIE PATON, F.R.C.S.

Patient, F.B., female, aged 18, an only child. Mother has had no miscarriages. No similar illness in the family.

Has been subject to frequent "colds," often with accompanying discharge from nose and ears, since infancy. No definite history of otitis media. Appendicectomy (April, 1920), followed by severe broncho-pneumonia and recovery. Otherwise healthy.

When she was aged 8 her parents noticed that she was unable to read small print, and vision gradually failed from that time until three years ago, when loss was complete (vision to each side being lost first—? bitemporal hemianopia). Menstruation, which has been regular and normal, began at age of 14 years, and from that time onward she has suffered from adiposity. Severe bilateral frontal headaches, mostly in evenings, for seven months. Recently hearing has occasionally been bad.

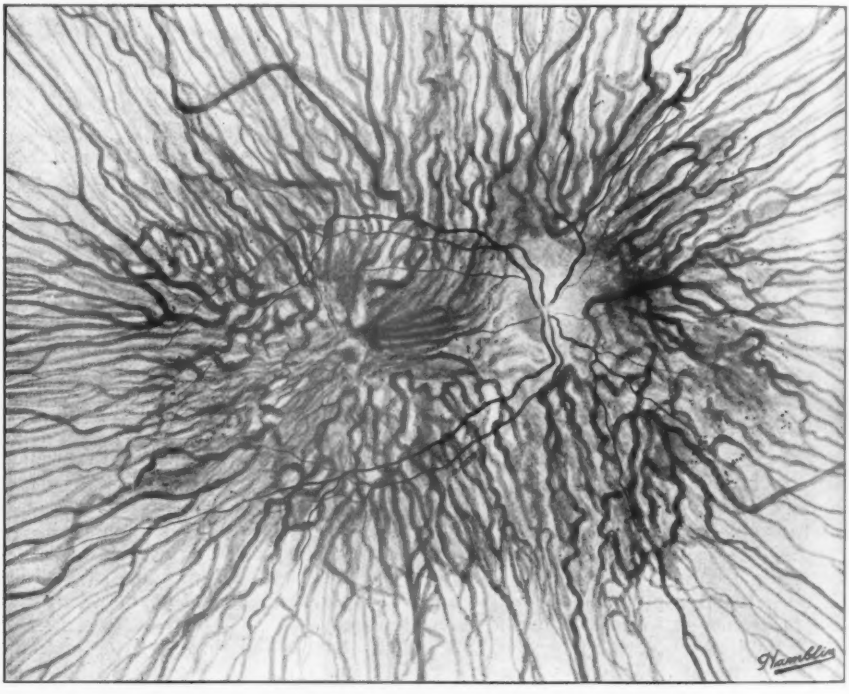


FIG. 1.—Atrophy of Retina and Choriocapillaris. (Mr. Leslie Paton's Case.)

Admitted to National Hospital, Queen Square, October, 1920, under Dr. James Collier.

Patient is of happy and bright disposition. Able to play three musical instruments. Speech and memory not defective in any way. Reads Braille easily and is able to knit. There is marked adiposity of Fröhlich type. Pubic and axillary hair normal in amount, scanty eyebrows.

Visual acuity: Cannot distinguish light from darkness.

Fundi (fig. 1): The condition is one of such complete atrophy of the retina that the whole course of the choroidal circulation can be seen in a diagrammatic fashion. In both fundi the only evidence of the existence of the retina is the fine threads of

retinal vessels and the few scattered pigment granules at the macula. The choriocapillaris has completely disappeared. Close to the macula one ciliary vessel can be seen to enter and spread out at once into a cluster of vessels. Farther out the long ciliary trunks can be seen passing forwards in parallel lines. In the presence of the more gross choroidal vessels the condition differs from the rare choroideremia.

Both pupils are small and regular and do not react to light. They dilate for a few seconds from time to time. No reaction on attempting to accommodate.

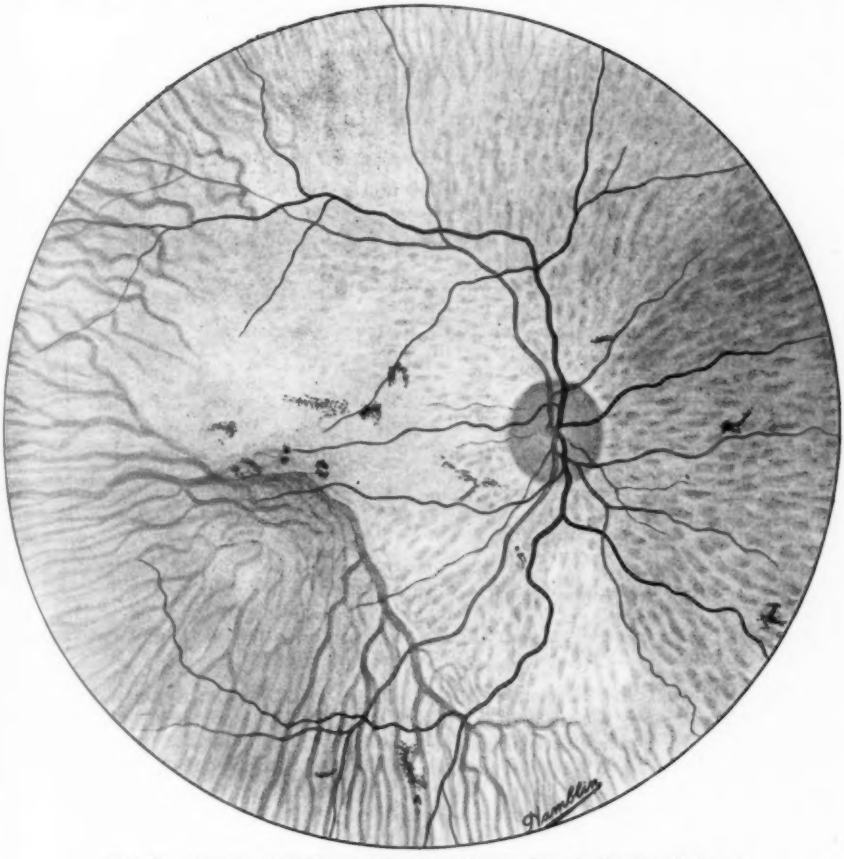


FIG. 2.—Atrophy of Retina and Choriocapillaris. (Mr. Leslie Paton's Case.)

No limitation of ocular movement. Coarse, irregular horizontal nystagmus on looking to right and left. The movements of the eyes are occasionally dissociated with strabismus, but the only regular feature of this is that the left eye tends to rotate laterally without accompanying movement of the right.

Some deafness in both ears, with occasional tinnitus on both sides; bone conduction greater than air conduction in both ears. Weber test referred to left ear. Gait steady. No other neurological abnormalities.

Evidences of an old pneumonic lesion on the left side. Blood-pressure 98/68. Normal blood-sugar curve. Skiagram of skull shows a normal sella turcica. Cerebrospinal fluid clear and colourless, 5 cells per c.mm. Total protein 0·04%. Lange no change. Wassermann reaction, negative, as also in blood.

This may be a case of pituitary tumour of the Fröhlich type, but the sella is normal, sugar tolerance is normal, and there are no neurological indications of pituitary disturbance.

The second illustration is that of a case which was also under the care of Dr. Collier. It will be noticed how similar the two cases are. In both there is an acquired atrophy of the choroid and retina. In the first the blindness began at 7 or 8 years of age, and progressed steadily; the patient now being absolutely blind. The other patient was not quite blind when I saw her in 1926. We have not been able to trace her since, and I do not know her present condition.

The patient, I. C., female, aged 18. Was admitted to the National Hospital in August, 1926.

History.—A series of fits three years earlier (ceased after two months). Ten months later giddiness, frontal headaches and general lassitude. During the last two years there has been increasing ptosis and tremor. During the last three years she has been noticeably mentally backward. Stiffness in right leg and unsteadiness in gait for three months.

Previous History.—Meningitis when aged 1 year and 9 months.

Family History.—Mother epileptic since age of 15 years. Father died of Bright's disease. Four siblings; others healthy. Three miscarriages.

On Examination.—Subnormal intelligence. No aphasia. Visual acuity: Right (fundus painting) recognizes objects at foot of bed. Visual acuity left $\frac{6}{36}$. Fields normal. Conjugate deviation to left, absent in left eye, restricted in right. Convergence restricted in right eye and absent in left. Ataxic gait and response to finger-nose test. No tremor. Wassermann negative in C.S.F. Blood-urea 37%.

Extreme atrophy of retina and choroid; the choriocapillaris has almost completely disappeared. Star-shaped patches of pigment (fig. 2). The condition has come to resemble the very rare choroideremia, but in this case is really secondary to an extensive specific chorioretinitis.

For comparison I show a picture of Mr. Usher's case of choroideremia [slide shown], and it can be seen how it differs from these acquired atrophies of the choroid and retina. It may be called congenital absence of choroid. There are only about twelve cases of it recorded altogether; and all have been in males. In my two cases of choroidal atrophy, the patients are females.

Membrane in the Anterior Chamber.—D. MAGOR CARDELL, F.R.C.S.

J. A., male, aged 36. Treated by Mr. J. H. Fisher for keratitis, 1914, 1919. Complains of "bright light hurting the eye."

On Examination.—The left eye shows on the back of the cornea, paracentrally, a lattice-work membrane composed of hyaline material with pigment lying here and there on and by the side of the strands composing the membrane. The membrane is closely applied to the posterior surface of the cornea, which has pigment deposited upon it in the vicinity. Below the lower part of the cornea are seen the "ghosts" of previously existing blood-vessels. There are signs of past iritis. The right eye shows signs of past iritis, but no hyaline membrane formation.

Retinitis in Diabetics.

By P. J. CAMMIDGE, M.D.

THE records of my last thousand cases of diabetes mellitus show that in 48, or 4·8%, the patients suffered from retinitis and retinal hæmorrhages as a complication. This proportion corresponds fairly closely to the 5·2% found in Joslin's cases in

America[1], but is considerably less than the 17% reported by von Noorden[2], and the 13% recorded by E. Grafe[3] for German patients. It is improbable that the higher percentage of cases exhibiting retinal changes in the German series is to be explained on diagnostic grounds, and I am inclined to think that the difference between the German and English or American figures is largely accounted for by the prevailing form of diabetes met with in these countries. In discussions on the complications of diabetes it is frequently forgotten that it is not a clinical or pathological entity, and that the cardinal symptom, glycosuria, may arise from a variety of causes. Neglecting the rarer forms, diabetes may, for practical purposes, be divided into two main types: (1) the anapothectic (or "alimentary") variety, in which there is difficulty in storing carbohydrate, and (2) the achriatic (or "true diabetic"), in which there is defective utilization as well as defective storage. As a rule, the former occurs in elderly people, while the latter usually develops before middle age, at least as a primary condition, and since diabetic retinitis is exceedingly rare before the age of 35, we should expect it to be generally associated with anapothectic glycosuria, which seems to be more common in Germany than in England or the United States, thus possibly explaining the German findings.

Published statistics, as I have mentioned, indicate that diabetic retinitis is rarely, if ever, met with in persons under 35 years of age; thus in 82 cases referred to by Nettleship[4] the youngest patient was aged 35, and in 62 cases described by Foster Moore[5] the earliest age of incidence was 39. My own figures show that in 47 out of the 48 cases the patients were aged 35 or over, the exception being a girl aged 19. This patient, who was only seen once in consultation, had suffered from diabetes, complicated by eczema, boils, and bleeding from the gums, for about two years: latterly she had complained of failing sight, and ophthalmoscopic examination had demonstrated a typical retinitis. Her blood, when I saw her, contained 240 mgm. per cent. of sugar, and analysis of the urine revealed 8·5% sugar, but no acetone or albumin. Her systolic blood-pressure was 240 mm. It is not possible to express a definite opinion as to the type of diabetes from which this patient was suffering, but judging from the available data and the fact that her maternal grandfather, her mother, and a maternal aunt, died from diabetes at the ages of 71, 49, and 48 respectively, which suggests that the disease was transmitted as a Mendelian dominant, it is probable that it was of the anapothectic variety. In this connection it is interesting to note that a family history of diabetes in immediate blood relations was obtained in 19 out of my 48 patients, suffering from retinitis, or approximately 40% compared with 28% in my diabetes cases as a whole. There is no evidence that the complication is related to sex, as the numbers were about equal (26 males and 22 females.)

When retinitis was first recognized in diabetics, by Edward Jaeger, about seventy years ago, it was naturally regarded as a direct effect of the disease; but, as experience accumulated, this explanation was gradually abandoned, and most authorities are now agreed that it is doubtful whether a retinitis due to diabetes alone ever occurs. One of the main reasons for this change of opinion is that there is not the relation between the severity of the diabetes and the incidence of the retinitis which would be expected if the retinal changes were a direct result of the metabolic disturbances resulting from the primary disease; on the contrary, retinitis occurs almost exclusively in patients with a mild and easily controlled glycosuria; thus Wagener and Wilder[6] found retinal disease in 38 out of 300 cases of diabetes

1 "Treatment of Diabetes," 4th ed., p. 736.
2 "Die Zuckerkrankheit," 1912, ii, p. 217.
3 *Klin. Woch.*, 1923, p. 1216.
4 *Roy. Lond. Ophth. Hosp. Rep.*, 1903, xv, 339.
5 *Trans. Ophth. Soc. U.K.*, 1920, xl, 16.
6 *Journ. Amer. Med. Assoc.*, 1921, lxxvi, 515.

of a mild type, but none in 80 cases characterized by acute onset and a progressively increasing severity. Confirmatory evidence is apparently supplied by the fact that retinal hæmorrhages occasionally develop in diabetic patients who have had no previous visual symptoms, some time after the urine has been rendered sugar-free, and the blood-sugar has been brought within the normal range, by appropriate treatment, as happened in four of my cases. The effect of insulin is interesting in this connection, for it has been my experience that, although the vision of patients with diabetic retinitis improves materially when their blood-sugar is controlled by suitable injections, the occurrence of retinal hæmorrhages is not appreciably influenced.

The frequent presence of albumin, as well as sugar, in the urine of diabetics suffering from retinitis, has suggested that the eye changes might be a consequence of concomitant renal defects. Writing in 1919, Onfray[7] stated that 25% of his cases had definite evidence of nephritis, while another 50% showed indications of some renal damage, and Garrod, at the Congress of the Ophthalmological Society in the following year,[8] declared that most of his diabetic patients with retinitis exhibited evidence of renal disease. Diabetics undoubtedly suffer from nephritis, and, as Schöbl pointed out in 1898,[9] there are cases in which the term " albuminuric retinitis in the eyes of diabetics " correctly describes the condition, but ophthalmologists appear to be agreed that there are typical differences between albuminuric and diabetic retinitis by which a diagnosis can be made in most instances on the ophthalmoscopic appearances alone. As a rule, the amount of albumin in the urine is small, unless the retinitis is of the typical albuminuric type, and in some instances none can be found, even when repeated examinations are made; thus Kako[10] reported albuminuria in only twenty-one out of fifty-seven cases, and in my series a faint trace of albumin was present in nine, and a measurable quantity in thirteen of the forty-eight cases. Undue importance should not be attached to the presence or absence of albumin in the urine, however, since it is well known that there is often little or no albuminuria in certain forms of renal disease. More reliable evidence of renal inefficiency can be obtained by special tests and by chemical analysis of the fasting blood, but very few investigations of this description appear to have been carried out in cases of diabetic retinitis. Wagener and Wilder,[11] using the phenolsulphonephthalein test in twenty-eight of their cases, found that only six of these patients excreted over 50% of the dye, so that in 78% there was apparently some deficiency of renal function, while estimation of the blood-urea in seventeen cases showed an excess in eight, or 47%. I have had the opportunity of making a detailed analysis of the blood in sixteen of my cases of diabetes in which the patients suffered from retinitis, and found that in nine of them there was distinct evidence of renal insufficiency, four showing an excess of nucleotide nitrogen, uric-acid, non-protein nitrogen, urea, and creatinine (+++++ Table I), and in five, although the creatinine was normal, there was an excess of the other end-products of nitrogenous metabolism (++++ Table I). In three other cases there was more or less nitrogen retention, one showing an excess of nucleotide nitrogen, uric acid and non-protein nitrogen (+++), another an excess of nucleotide nitrogen and uric acid (++), and the third only an excess of nucleotide nitrogen (+), and as the urine of all three contained traces of albumin, it is probable there was some renal insufficiency. Four cases gave normal figures for all the nitrogenous waste products in the blood, however, and showed no trace of albumin or other evidence of renal disturbance in the urine, so that in 25%, at least, the retinal changes were apparently not associated with defects in the functions of the kidneys.

7 *Ann. d'ocul.*, 1919, 155, 533.
8 *Trans. Ophth. Soc. U.K.*, 1920, xl, 6
9 Norris and Oliver, " System of Dis. of the Eye," 1898, pp. 3 and 413
10 *Klin. Monat. für Augen.*, 1903, xli, 257.
11 *Op. cit.*

The other results of the analyses made in these cases showed a characteristic hyperglycæmia in all but one, twelve having an excess of sugar in the blood, fasting and after a test meal (++ Table I), and three only after the test meal. (+ Table I). The exception was a patient who was sent to me with a diagnosis of "diabetic retinitis," made by an ophthalmic surgeon who had been consulted a few weeks previously. Sugar was stated to have been found in the urine at intervals during the preceding six years, but when the patient came to me none could be discovered, nor did any glycosuria or hyperglycæmia develop during the time she was under my care, in spite of the fact that a considerable amount of carbohydrate was eventually allowed. More or less sugar was found in the urine of all the other patients, although in two instances it had diminished to a trace too small for accurate determination, some time before the eye complication developed. Judging from the percentage of lactic acid in the blood, five of the patients in which it was low had distinct difficulty in utilizing carbohydrate, and, as three of these showed an excess of acetone in their urine, there was evidently difficulty in fat utilization as well, but the normal level of the lactic acid in eleven indicated a simple anapothectic glycosuria, due to defective storage of carbohydrate. Slight salt retention was found in the fasting blood of two of the patients, and an excess of phenols, indicating toxic absorption, was met with in twelve out of the sixteen. Apart from the hyperglycæmia, the most constant abnormality of the fasting blood in these cases was the low level of calcium, thirteen, or 81%, giving figures which were distinctly pathological, and as a similar condition was found in only 44% of the total series of diabetics, it would seem justifiable to infer that this abnormality is one of the factors concerned in the production of the complication from which they suffered. This is even more likely if we exclude the three patients with a normal percentage of calcium in their blood, as we probably can, for they were all apparently examples of albuminuric rather than diabetic retinitis, since they showed advanced nitrogen retention and passed a large amount of albumin in their urine.

A deficiency of calcium in the blood may arise from a variety of causes, but in diabetes the most common is depletion of the alkali reserve of the body consequent on acidosis. This was the explanation in exactly one half of my general series of diabetics, but since only four of the patients we are considering were found to be passing acetone in their urine, or to show an excess in their blood, it cannot account for the condition in more than a small proportion, at most, of those suffering from retinitis. Normally, calcium is excreted mainly by way of the large intestine, and in chronic inflammatory affections of the bowel there is a characteristic increase; it is interesting, therefore, to see that in eight of the cases there was an abnormal percentage of calcium in the stools, but as one of the patients had a normal blood calcium, and six of them with a low blood calcium were found to have a normal percentage in the fæces, excessive loss through the intestinal wall is evidently not a constant ætiological factor. Again, although an abnormal excretion of calcium in the urine occurred in nine of the cases, combined with an excess in the fæces in five, it was not a characteristic feature of all the cases. In two instances a low blood calcium was associated with a normal excretion of lime salts in both the urine and fæces. Recent investigations by Morrison and Bogan [12] suggest another possible explanation of the deficiency of calcium in the blood of elderly diabetics. As a result of the roentgenographic study of the feet and legs of a large number of cases these observers found that, as in normal persons, calcification of the vessels is rare before the age of 40, although after that age the proportion showing calcification, and also the degree of calcification, increased much more rapidly in the diabetic than the non-diabetic; thus, in normal persons between the ages of 40 and 50 the vessels of the legs showed evidence of calcification in 36%, whereas in diabetics of the same decade 63% were similarly affected, and of these 5% had suffered from

12 *Journ. Amer. Med. Assoc.*, 1929, xcii, 1424.

glycosuria for under four years, 50% were known to have had diabetes for five to ten years, and 75% for over twenty years. According to Borchardt [13] the pathological changes in the vessels differ in no way from those found in ordinary senile arteriosclerosis, and as the lesions closely resemble those produced experimentally in rabbits by prolonged feeding with cholesterol, it seems likely that persistent lipæmia, which is found with increasing frequency in elderly diabetics, with the passage of time, is the determining cause of the changes in the vessel walls. Recent investigations by Wade on thyroparathyroidectomized dogs [14] have indicated that there is a relation between the metabolism of cholesterin and calcium, a fall in the blood calcium usually corresponding with a rise in the cholesterol, and vice versa. We should therefore expect a high cholesterol to be associated with the low blood calcium in diabetic retinitis. Unfortunately the cholesterol content of the blood has been determined in only six of my cases; in four it was high, but in two there was a normal percentage. It may be a coincidence, but it is rather remarkable that the former were all typical cases of diabetic retinitis and showed no excess of calcium in the fæces, while the two with a low calcium and a normal cholesterol developed retinal hæmorrhages without previous visual symptoms at a time when the urine was free from sugar, and both exhibited an excess of calcium in the stools and urine.

Whatever may be the exact cause, or causes, of the calcium deficiency of the blood in diabetic retinitis, there can be little doubt that the diminished coagulability to which it gives rise, is one of the factors, and possibly the most important factor, in the production of the local recurring hæmorrhages which are of such grave prognostic significance. In some instances at least there is, however, probably a contributory factor. Onfray appears to have been the first to draw attention, in 1919, to the frequent association of vascular hypertension with diabetic retinitis, and it is now generally recognized that a systolic blood-pressure of 150 mm. or more is usually found. Occasionally much higher readings are met with, but in my experience a very high blood-pressure is rare unless there is much albumin in the urine and considerable nitrogen, when the condition is actually an albuminuric retinitis rather than a diabetic retinitis. One of my cases which falls into this category (No. IV) had a blood-pressure of 280 mm.; eventually the patient died from cerebral hæmorrhage, and it is interesting to note that this was one of the three cases in which a normal percentage of calcium was found in the blood. The average systolic blood-pressure in my series was 174 mm., but in four instances it was under 150 mm., and in one of these was only 130 mm., a normal figure for the age of the patient, which was 38 years. Foster Moore [15] states that on several occasions he has seen diabetics between the ages of 40 and 50 with retinitis in whom the blood-pressure was not at all raised, and the urine was free from albumin on repeated examination, and Chauffard [16] quotes the case of a man aged 68 with no hypertension or renal insufficiency in which immediate improvement followed treatment with insulin. It is evident, therefore, that although an abnormally high systolic pressure may be a contributory cause of the hæmorrhage met with in diabetic retinitis, it is not an essential factor, as the deficiency of calcium appears to be. Since diabetes of itself does not give rise to hypertension, it must originate in those cases of diabetic retinitis in which it occurs from some other cause, and as Morrison and Bogan found that patients with calcified vessels who were 50 years of age or over were nearly twice as apt to have a high blood-pressure as those without calcification, it would seem probable that the tendency to hypertension, like the deficiency of calcium, is to be attributed mainly to the metabolic defects of a senile organism rather than to the direct effects of the diabetic condition itself.

13 *Virchow's Arch. f. Path. Anat.*, 1926, cclix, 521.
14 *Amer. Journ. Med. Sci.*, 1929, clxxvii, 790.
15 *Medical Ophthalmology*, 1925, p. 203.
16 *Compt. Rend. Soc. Biol.*, 1925, xcii, 1356.

The rapid improvement in the vision of many patients suffering from diabetic retinitis, which follows when the hyperglycæmia and glycosuria are controlled by treatment, in spite of the fact that the tendency to local hæmorrhages persists, suggests that the changes interfering with visual perception and the hæmorrhagic tendency are, to a considerable extent, at least, of independent origin, the former being apparently dependent upon alterations induced by the diabetic state, whereas the latter, as we have seen, arises from defects which are not a direct result of the metabolic disturbances induced by the diabetes. Such local changes are probably the œdema of the retina and the hyaline and fatty changes found microscopically by Nettleship [17] and others. The œdema is probably a result of alterations in the osmotic pressure in the tissues caused by the presence of an excess of sugar in the blood, while it is well known that fatty and hyaline degeneration are of frequent occurrence in other situations in long-standing cases of diabetes, owing probably to defects of nutrition. When the carbohydrate metabolism of the body is improved, either by limitation of the diet alone, or with the help of insulin, and the sugar content of the blood is maintained within the normal range, the œdema no doubt subsides and the lipoid deposits are absorbed, with consequent improvement in the vision of the patient, but, as the treatment has no effect upon the calcium content of the blood, the danger from local hæmorrhages persists. The discovery of retinal hæmorrhages without previous symptoms, in the eyes of diabetic patients who have been sugar-free for long periods, extending in one of my cases to over two years, is thus accounted for, and tends to emphasize the independent pathology of the two conditions.

If the conclusions I have suggested are correct, it follows that the diagnosis and treatment of the retinitis occurring in the eyes of diabetics are not such simple problems as they are often assumed to be. Examination by the ophthalmoscope is no doubt the first step in the diagnosis, but, although I acknowledge with admiration the skill of the expert ophthalmologist, I am inclined to think that an accurate differentiation of the various types can only be achieved by chemical analysis of the blood under test conditions. In this way it is possible to determine with considerable certainty the functional efficiency of the kidneys, as well as the nature and extent of the defect of carbohydrate metabolism, and, at the same time, discover whether special treatment to counteract the effect of a deficiency of calcium in the circulation will be required.

I do not propose to enter into a detailed discussion of the treatment of diabetes, or of the modifications necessary when there is nitrogen retention and renal insufficiency; it will be sufficient if I point out that, as the glycosuria met with in most cases of diabetic retinitis is of the simple anapothectic variety, suitable quantitative regulation of the diet is usually sufficient to bring the blood sugar within the normal range and provide the necessary opportunity for recovery and repair. In the more severe cases, in which the use of insulin is advisable owing to the presence of defects of utilization and of secondary defects of metabolism, it is wise to commence with small doses and thus avoid a too rapid reduction of the blood sugar, since the sudden changes in the osmotic pressure which result from large doses may give rise to very disquieting symptoms. Appropriate dieting and attention to the condition of the alimentary tract will generally improve the functional activity of the kidneys and often bring about a considerable reduction in the blood pressure, so that the liability to local hæmorrhages is diminished, but if the calcium content of the blood is low, this danger persists, and may have disastrous consequences unless steps are taken to deal with it. As a palliative, calcium salts given by the mouth seem to be quite efficacious, although they have to be continued indefinitely. I usually find that 5 gr. of calcium lactate once or twice a day are sufficient to maintain the percentage of calcium in the blood within the normal range and prevent further hæmorrhages. Lately I have been experimenting with

[17] *Ophth. Hosp. Rep.*, 1879, vol. ix.

parathyroid and with vitamin D, but my experience of both is as yet too limited to permit of a definite opinion regarding their use in these cases.

As an example of the benefit of calcium therapy in a case of diabetic retinitis, I will quote the case of a man, aged 71, who was sent to me by Dr. Brockwell, of Walton-on-Naze. Sugar had been found in his urine accidentally in 1910, but, as he had no symptoms, he had not taken the matter seriously and paid no particular attention to his diet, except for a brief interval in 1924, when he was operated on by Sir Arnold Lawson for cataract. In the summer of 1927 he again consulted Sir Arnold Lawson for failing vision, and a characteristic retinitis with small hæmorrhages in the fundus of both eyes, was then discovered. When he came under my care in September, examination of his urine showed 2·6% of sugar, with a total of 44 grm. for the twenty-four hours, and a trace of acetone (0·7 grm.), but no albumin. The sugar content of the fasting blood was found to be within the normal range, 93 mgm. per cent, but after a test breakfast containing 40 grm. of carbohydrate, a mild hyperglycæmia developed, the maximum figure reached being 200 mgm. per cent. in one hour. Analysis of the fasting whole blood showed a slight excess of acetone (0·4 mgm.), a rather high percentage of phenols (7 mgm.), and a subnormal calcium (5 mgm.), but normal figures were obtained for the end-products of nitrogenous metabolism, and for lactic acid. The fæces gave a marked reaction for biliverdin, indicating excessive peristalsis in the upper intestine, and showed evidence of acid fermentation, while a high trypsin index and low percentage of fat pointed to the existence of a catarrhal pancreatitis. A blood-count revealed a slight anæmia, 4½ million erythrocytes and a hæmoglobin of 65%, which was possibly accounted for by the presence of a considerable amount of occult blood in the stools. Taken as a whole, these results suggested that the patient was suffering from an anapothectic glycosuria of long standing, which had progressed to a stage at which defects of sugar utilization had commenced, and these in their turn were giving rise to early faults of fat metabolism. In all probability the condition was partly hereditary, for there was a history of a paternal uncle having died of diabetes, but the exciting cause appeared to be a chronic catarrh of the upper intestine with secondary infection of the pancreatin and bile-ducts. Treatment of the intestinal catarrh and physiological rest of the alimentary tract brought about a rapid improvement in both the local and general conditions, but, as it was eventually found impossible to provide an adequate diet without hyperglycæmia and glycosuria, the patient was given a small dose of insulin, 8 units twice daily. The subsequent progress of the case was uneventful as far as the diabetes was concerned, the blood sugar ranging between 80 and 150 mgm. per cent., and the urine remaining free from any trace of sugar or acetone, but the state of the patient's eyes still continued to cause concern. Sir Arnold Lawson, writing on May 17, 1928, said: "I am glad to see the treatment has had a good effect on the sight. The patient has recovered nearly all the lost ground and can now read the smallest diamond type with a little trouble, but his condition is not altogether satisfactory, for he is still getting hæmorrhages in the fundus, and they portend ill for the future." A further examination of the patient's metabolism was then made, and it was discovered that (1) the calcium content of the blood had fallen to 4·5 mgm. per cent.; (2) the excretion of calcium in the urine had risen from 1·2 to 2·6 grm. a day, and (3) the blood-pressure, which had stood at 148 mm. in September, was now 158 mm. Calcium lactate, in 5-gr. doses three times a day, was accordingly prescribed, to make good the deficiency in the blood, and the diet was readjusted with a view to bringing down the blood-pressure. A month after the commencement of this treatment a blood-pressure of 137 mm. was found, and examination of the blood showed 6 mgm. per cent. of calcium. A month later the blood calcium had risen to 6·5 mgm. per cent., the maximum normal figure. After an examination of the patient's eyes in August, Sir Arnold Lawson reported: "there are no fresh hæmorrhages, and there have evidently been none for some time." An

attempt was then made to discontinue the calcium, but as the percentage of calcium in the blood rapidly fell, and fresh hæmorrhages made their appearance, it was resumed, although smaller doses of 5 gr. a day were ultimately found to be sufficient to maintain the percentage in the blood within normal limits and prevent fresh hæmorrhages.

Other cases have shown similar results and demonstrated that the administration of lime salts by the mouth in sufficient amount to maintain the percentage of calcium in the blood at a normal level, prevents local hæmorrhages in diabetic retinitis, which is, I think, conclusive evidence that a calcium deficiency is an essential factor in the complication. The occurrence of hæmorrhages in diabetics with advanced renal disease, when there is no calcium deficiency in the blood, is, on the other hand, not influenced by treatment with calcium salts.

RETINITIS IN DIABETICS.

Case number	Sex	Age, years	Heredity	Duration, years	Urine				Blood								Fæces			Blood-pressure, mm.
					Sugar, %	Acetone	Albumin	Calcium	Sugar	Lactic acid	Acetone	Nitrogen	Phenols	Salts	Magnesium	Calcium	Billverdin	Ash	Calcium	
I	M	38	+	2	10	++	0	+	++	L	++	N	+	+	N	L	+	+	+	130
II	F	54	+	7	4	0	0	N	++	L	N	++++	N	N	N	L	N	N	N	206
III	M	57	−	10	7	0	+++	N	++	N	N	++++++	+	+	N	N	N	+	+	190
IV	F	58	+	28	3	0	+++	N	+	N	N	++++++	+	N	N	N	+	N	N	280
V	F	60	+	5	0	0	0	+	N	N	N	++++	+	N	N	L	+	+	+	160
VI	M	62	+	14	2	0	tr	+	++	N	N	+++	N	N	N	L	+	+	+	148
VII	M	64	+	10	1	0	+++	N	++	N	N	++++++	+	N	N	N	+	+	N	165
VIII	F	65	−	9	1	0	0	+	++	L	N	N	N	N	L	L	+	+	+	180
IX	F	65	−	10	2	+	tr	+	++	L	+	++	N	N	N	L	+	+	N	148
X	M	65	−	17	tr	0	0	+	+	N	N	N	+	N	N	L	+	+	+	158
XI	M	67	+	7	2	0	+	N	++	N	N	++++++	+	N	N	L	+	+	+	172
XII	F	67	+	6	tr	0	tr	+	++	N	N	++++	+	N	N	L	+	+	N	156
XIII	F	68	−	15	4	0	+	N	++	N	N	++++	+	N	N	L	+	+	N	160
XIV	M	70	+	10	5	+	tr	N	++	N	+	++++	+	N	N	L	+	+	+	180
XV	M	71	+	17	4	0	0	+	+	N	N	N	+	N	N	L	N	N	N	148
XVI	F	73	−	4	6	++	tr	+	++	L	+	+	+	N	L	L	+	N	N	198

Discussion.—Dr. R. D. LAWRENCE said that he regarded diabetic retinitis as an unexplained mystery. He had looked through the accounts of cases which had been treated at King's College Hospital, and had attempted to see what factors, other than the diabetes, might be behind the production of the eye condition.

In the first place these cases showed a considerably higher incidence than Dr. Cammidge's (about 10%). Probably retinal changes were actually more common than this in his series, as the eyes were usually not carefully examined (i.e., by the ophthalmologist) unless defective vision was complained of. The retinitis occurred chiefly in elderly patients; only one had been under 50. Albuminuria, high blood-pressure and general arteriosclerosis were common, but cases occurred in which there was no evidence of these factors. In cases with no evident arteriosclerosis, he imagined that the cause of the exudates and typical hæmorrhages was intimal degeneration in the small vessels and capillaries. This was the typical damage found in diabetic arterial change throughout the body, and no doubt it occurred in the retinal vessels as well. Another factor was possibly constituted by the changes in osmotic pressure of the blood, accompanying varying degrees of hyperglycæmia, but this was probably only a small factor in retinitis, although very important in diabetic cataract.

Dr. Cammidge's suggestion of a low blood calcium as a causal factor was a completely new idea to him (the speaker) and was contrary to his experience in diabetic cases.

Colonel A. E. J. LISTER asked in what proportion of cases the retinitis cleared up. He also asked how much calcium was given to the patients, and in what form. His experience in India, where the disease was very common, was that the proportion of diabetics with retinitis was higher than that which Dr. Cammidge gave; it approached more nearly to the German figure. The age-incidence was also somewhat younger in India, where people aged more quickly than in this country.

Dr. CAMMIDGE (in reply) said that Dr. Lawrence spoke of hospital cases which were generally of long standing, but his (the speaker's) had been private cases which were usually seen at an earlier stage. He had only quoted cases as "retinitis" which had been sent to him as such. He did not profess to be an expert in the examination of eyes; and, though he saw a number of patients who complained of misty vision, he would not say they had retinitis; in any case it had not been sufficiently serious to impel them to see a doctor or an ophthalmic surgeon. That all his cases were typical ones might be another reason why his percentages were low.

It was difficult to come to a conclusion as to the ætiology of the condition. He had discussed the matter fully in his paper. He was inclined to agree with Dr. Lawrence that the retinitis was not altogether due to the sugar, but he did think that the œdema, etc., which interfered with vision, were due to sugar, because the condition cleared up so rapidly under treatment, especially now that the patients could have insulin.

The only other question asked concerned treatment. The people who had this retinitis were generally elderly or old, and he had a strong objection to giving insulin to old people if it could be avoided. These patients had already a certain amount of change in their heart muscle, and if the amount of sugar going to the heart were quickly interfered with, the consequences might be very serious. His view was that if these elderly diabetics could be treated by dieting alone they were much more comfortable, and they did quite well, though it might take longer to bring about a satisfactory result. In some of the cases one felt bound to use insulin, but it was necessary to watch carefully the reduction in the quantity of sugar. In his work it was made a matter of routine to estimate the calcium in the blood, just as the sugar in the blood was estimated. In all these cases a low calcium content was found, though the routine was not carried out with that object. It was not until the results were classified that the low calcium content was discovered. As to the treatment, he generally gave calcium lactate, but as most of the patients had some intestinal catarrh, he gave the calcium in an emulsion of paraffin. A beginning was made with 5 gr. three times a day. The calcium in the blood was estimated, and when it reached the normal amount, it was generally found that the patient could do with 5 or 7 gr. a day. This was, however, only a prophylactic and it was necessary to continue it for the rest of life. The important point, so far as he himself was concerned, was that the ophthalmic surgeon who only treated his cases of diabetic retinitis for diabetes was not likely to do his best for the patient; he must also treat the patient for calcium deficiency when that was present, and so prevent his having the recurrent hæmorrhages, which were of such grave prognostic significance.

Two Sterilizers for Ballantyne's Drop Bottles.—J. W. TUDOR THOMAS, F.R.C.S.—The first of these has perforated sides and is intended for hospital use in a steam container. The other is a simple sterilizer for use at home, the sterilizing being carried out at the temperature of boiling water. It also forms a portable and sterile carrier for the drop bottles and their stand, which are not touched by the fingers until required for use.

Prismatic Spectacles.—J. W. TUDOR THOMAS, F.R.C.S.—These were made for a case of spondylitis deformans, in which kyphosis of the spine was so marked that the patient could not see more than half way up a person standing in front of him and, when taking a walk, could not see the surrounding objects at all.

The glasses consist of prisms of 20 prism dioptres, fitted base up, with the base close to the eyebrow, and the apex pointing to the ground. The lens measures 40 mm. by 16 mm., and is rimless. The bridge is carried back and down by two rigid supports. When the spectacles are fitted, the patient has an uninterrupted view of the ground for three yards ahead, and by glancing upwards he can recognize his friends in the street, avoid approaching traffic, and enjoy the surrounding scenery.

Section of Orthopædics.

President—Mr. D. McCrae Aitken, F.R.C.S.

[*November 5*, 1929.]

PRESIDENT'S ADDRESS.

Respiratory Rhythm in Physiological Relation to Movement and Posture.

By D. McCrae Aitken, F.R.C.S.

In searching for a suitable subject for my presidential address, I felt that it ought to deal with the ground-work of orthopædic practice, should be in some measure an academic thesis reflecting some, at least, of my personal faith derived from over twenty years of orthopædic work, and might, perhaps, present some of the simplest facts of common experience in a fresh light so as to stimulate thought or provoke investigation by others.

The theme I shall try to put before you is that :—

(1) Every posture of the body has a definite natural association with respiratory movement.

(2) Most, if not all, of the natural purposeful movements of the body which we use in standing, walking, running or working with our hands and arms are most easily made at a definite time in the respiratory cycle of inspiration and expiration.

(3) In patients in whom the natural use of the body as a machine has been put out of order by paralysis, injury or disease, the restoration of natural automatic movement is made much more easy if the patient be first taught to make the difficult movement in proper rhythm with respiration.

(4) Finally, a correct relation between respiration and posture materially assists the heart's action, helps to support abdominal viscera and promotes the well-being of the whole body.

The way in which I now think of movement and respiration as essentially coördinate has been arrived at by the convergence of several distinct lines of observation and thought.

First, more than twenty years ago, Sir Robert Jones urged me to try to bring the treatment of scoliosis into line with the treatment of other deformities, such as club foot, knock knee, or dropped wrist, which give rise to defects of posture and movement, the essential principle of treatment being to retain the distorted limb in a corrected position, if necessary by a splint, until the coördinate reflex balance between the several groups of muscles is naturally restored, while adaptive growth-changes also help to adjust the machine. At Manchester, I showed a film to demonstrate how the essential relation between posture and respiration can be used in the treatment of scoliosis. I now show this film to demonstrate the relationship which exists between posture and respiration.

The second line of thought which has been running in my mind, also, for twenty years, was started by the work of Magnus and Sherrington in decerebrate dogs and cats. On this work the modern doctrine of the postural reflexes has been based, Magnus being, I think, the originator, and Sherrington one of its most active supporters in this country. Magnus, working first in Liverpool and subsequently in Utrecht, set himself to study reflexes in decerebrate dogs and cats. The gist of his work was that, e.g., in the dog, if one hind limb be flexed and the other paw tickled, the flexed limb reflexly extends, while, if the limb be first placed in an extended position, the reflex action is flexor. Similar results were obtained in experiments on the muscles of the cat's tail. Magnus, therefore, concluded that there was some determining factor in the posture of the muscle which was quite independent.

When I read this work about 1908, the idea intrigued me in relation to spastic paralysis, for the method of attack I had learned from Robert Jones was to disarm the spastic muscle by overstretching it, or by dividing its tendon, and then to restore coördinate movement by training. Since that time others have introduced operations on peripheral nerves, posterior roots and sympathetic nerves, in order to cut the reflex cycle in spastic paralysis. Now, if Magnus' original conclusion be correct—that there is something in the posture of the muscle which decides its readiness to react to reflex stimuli—all such procedures may be mistaken in their physiological basis.

Thirdly, many of you are aware that respiratory difficulties have beset my own vile body, and I have had to learn to do my own work with economy of breath and the minimum of back-pressure on the heart. Personal experience has, therefore, been part of my school, and in recent years I have been trying to apply some of the lessons I have learned from my own body to the restoration of easy movement in others—sometimes with gratifying results.

In what follows, I wish to demonstrate that there is a clinical physiological connection between respiration and those multiple coördinate actions of muscles which we employ in every ordinary automatic movement, such as playing games, playing musical instruments or handling tools. Further, the study of this clinical physiology has hardly been touched by our profession. To give examples familiar to all of us; when you or I stoop to pick up a heavy bag, as we lift it we automatically take a breath. In-breathing seems to have a natural association with lifting. If this be true, then the reverse group of movements should be associated with out-breathing. Consider, therefore, the action of sculling a skiff. Pulling the sculls through the water means extending the lower limbs and trunk, and pulling with the upper limbs against resistance. This is comparable to weight lifting, and is an inspiratory effort. Coming forward is done in time with expiration, with open glottis. The abdominal muscles contract to flex the trunk and drive the abdominal viscera up against the diaphragm, and help to expel air from the lungs. Those who are familiar with Goldthwait's postural exercises, or who heard the address he gave in July, 1929, will remember that proper use of abdominal muscles to initiate expiratory movement is a fundamental part of his teaching. In rowing, the arms shoot forward at the same time as the legs relax and the trunk is flexed. Extensor or pushing movements of the upper limb should, therefore, be associated with expiration. Consider the act of driving a large screw nail into hard wood. Those of us who have done this must have frequently caught ourselves giving an expiratory grunt as we push and supinate, which suggests that forcible supination of the forearm, as well as pushing, is intimately associated with the expiratory phase in respiration; it is impossible to drive a screw hard during inspiration. To test this point, consider next a familiar movement in which the left hand grips the tool, namely: the act of driving a golf ball. The shaft of the club is gripped by the third, fourth and fifth fingers of the left hand, mainly with the fourth and fifth; the "follow through" must be done with a supination movement of the left forearm, whether in an approach shot or a full drive. I suggest that the fault of "pressing" is most often due to closing the glottis and so fixing the thorax, instead of letting the air steadily pass out during the completion of the movement. Inspiration is ordinarily associated with raising the head. "Lifting the head" is the term applied to a common fault at golf, which again I believe to be really a sudden closure of the glottis interfering with a perfect "follow through." Strokes at tennis, leading in boxing, or lunging in fencing, all have definite association with breathing, if done well.

To digress for a moment, the association of inspiration with gripping and pronation, and of expiration with supination and with extending the fingers, has been tried out with encouraging results in re-educating hand movements in cases of spastic diplegia. The patient must, of course, first be taught to breathe easily and regularly, and this is often the more difficult half of the battle.

About two years ago, one of my gymnasts pointed out that she could discover no association between breathing and movements of the thumb. In monkeys, of course, the hand is an organ of progression in arboreal life; the spider monkeys have no thumb, and swing along very skilfully without. Man being a completely erect animal, has emancipated his hand from taking part in ordinary progression, but has retained some of its postural associations in connection with pulling, climbing trees, getting on to 'buses, and so forth, and also conversely with pushing. For the rest, the hand has been set free to acquire a host of new automatic reflexes, such as writing, stenography from dictation, playing musical instruments or games in which automatic reflexes follow afferent impulses from ear or eye, and are quite dissociated from any respiratory association as they usually are not performed against the force of gravity or other resisting force. If it be true that the thumb is emancipated more than the rest of the limb from association with more primitive reflexes, it may be one more point in the evidence that the possession of a thumb is a factor in the assumed superiority of man in the animal kingdom.

The ideas I have just put before you came to me through working back over ground which I have covered in trying to treat scoliosis. We can and do use the limbs in two entirely distinct ways. For example, if I sit on a table and swing my legs to and fro, I am, doubtless, using the flexor and extensor muscles of my knees, but such movement has no relation with the purposeful or weight-carrying function of muscle. Such movements never have been—and never will be—used for the purpose of driving a machine. On the other hand, driving the treadle of a turning lathe, or the pedals of a bicycle comes into relation with the ordinary static or walking function of muscles, is associated with our ordinary postural reflex mechanism and can be carried on easily without conscious effort, and therefore without fatigue.

In this connection the usual anatomical description of the origins and insertions of muscles, and of their actions, cannot satisfy the orthopædic surgeon. The anatomist describes the body as he sees it lying on a slab; to him the lower limb is an appendage of the trunk. The orthopædic surgeon's view of the body is biological, not necrological; to him the body stands vivid and erect on the feet. The lower limbs are not appendages of the trunk, they are the living pillars on which the whole body is carried and balanced. Man lives and moves on his feet; the muscles which control his balance work from below upwards. At any rate with regard to the lower limb, most of the accepted descriptions of origins and insertions are upside-down, and the action of the muscle usually described is topsy-turvy with regard to the manner in which the muscles normally act in maintaining posture and in locomotion. The tendo Achillis must be regarded as the common tendon of origin of the soleus, which helps to balance the tibia on the ankle-joint, and of the gastrocnemius which prolongs a similar action to above the knee. The ligamentum patellæ is the common origin of the two vasti and the crureus muscles, which control the knee, and the rectus femoris, which posturally is a flexor of the hip-joint serving mainly to balance the pelvis. I think this last statement is correct, for I injured this muscle in my own thigh, and then discovered that lying on a table I could fully extend the knee without pain, but could not bend forward to field a ball, or perform any active flexion of the hip without pain. I therefore believe that this muscle in its postural function is mainly a flexor of the hip, and only indirectly an extensor of the knee; observations by others are, however, required to check such a suggestion. One other muscle must be mentioned in this connection, namely, the tranversalis abdominis, which forms a continuous sheet with the triangularis sterni muscle above. In textbooks of anatomy the triangularis sterni is correctly described as rising from the back of the sternum and being inserted into the ribs as high as the second. The transversalis abdominis is described the wrong way round, it should be described as rising from the crest of the pubis and the linea alba, and being inserted into the inner lip of the crest of the ileum into the lumbar vertebræ, by the

several layers of the lumbar aponeurosis, and into the six lower ribs in a manner similar to the insertion of the triangularis sterni, which is the upper part of this great muscle of expiration. This muscle helps to support the thorax on the pelvis; it is the muscle which should initiate the act of expiration; its lower fibres support the abdominal viscera against the under surface of the diaphragm, while its upper portion pulls down the ribs, narrows the outlet of the thorax, and allows the dome of the relaxing diaphragm to rise still further against the collapsing lungs. Of course, in actual practice the other muscles of the abdominal wall are associated with the transversalis abdominis. All these muscles actively participate in both the static and respiratory parts of our postural reflexes. Further, muscles of the thighs and legs must act synergically to steady the pelvis while the abdominal muscles do this work. The transversalis muscle, with its associates, is the muscle by which the singer gets that first clear prompt note, technically known as the attack; the boxer and the rowing man both use it: in boxing all punching forward, with the trunk moving forward, is done in expiration, if done well. The regular rhythmic action of this muscle with associated action of the pelvic floor assists the return of the blood from pelvis and abdomen, and its use should have its due place in the treatment of backaches, especially those associated with pelvic congestions in women. Talking, reading, writing, even our manners, become automatic reflex actions by constant practice; but I think it is only those group actions of the muscles which are associated with physical effort which have acquired a definite association with respiration, perhaps because a correct rhythm assists the return of blood by the venæ cavæ and is economical of cardiac effort.

It must be admitted that there are exceptions to automatic learning even o ordinary movements. The deaf mute does not learn to talk, because there is a flaw in the reflex nervous cycle, but he can be taught by lip reading. The child with a congenital spastic paralysis may not learn to walk, but if he can be brought into an erect posture with the soles of his feet to the ground, he may be able to acquire balance reflexes and to learn to walk, although his reaction times remain slow. All athletic training is really training of nerve reflexes, inhibition being more important than contraction of muscle, for it quickens the reaction time and makes for alertness and delicacy of coördination. The theme I have put forward is that there is a natural reflex association between respiratory rhythm and the coördinate group actions of muscles, which we employ in the ordinary actions of life, and that these reflex associations might well be utilized in efforts to re-educate defective function.

Results obtained so far, and the support from gymnasts and after-care sisters who have helped me to try out these ideas, are encouraging. The ground has not been fully explored, and I am prepared to find that any or all of my suggestions are open to correction. I hasten to put them forward, even in an immature form, in order that others in our profession interested in this work may discover what degree of usefulness they may possess.

The ideas I have been putting forward have been arrived at by working backwards over ground I have traversed in the last twenty years in the treatment of scoliosis.

* * *

[The remainder of the address took the form of a running commentary on lantern slides and moving pictures, which showed how varying posture and respiratory movement reacted on each other in scoliosis. The film was prepared to demonstrate the principles of treatment of scoliosis. As it is impossible to put in print a description of the ripple of associated muscles which can be seen in a moving picture, all that can be done here is to recapitulate briefly the points demonstrated by the writer.

First, scoliosis is not merely a deformity of the vertebral column. It is an asymmetrical twisted deformity of the whole trunk, involving asymmetrical distribution of weight on the feet, which involves asymmetrical action of balance muscles, and of respiratory muscles. Thus a patient with primary defect of the

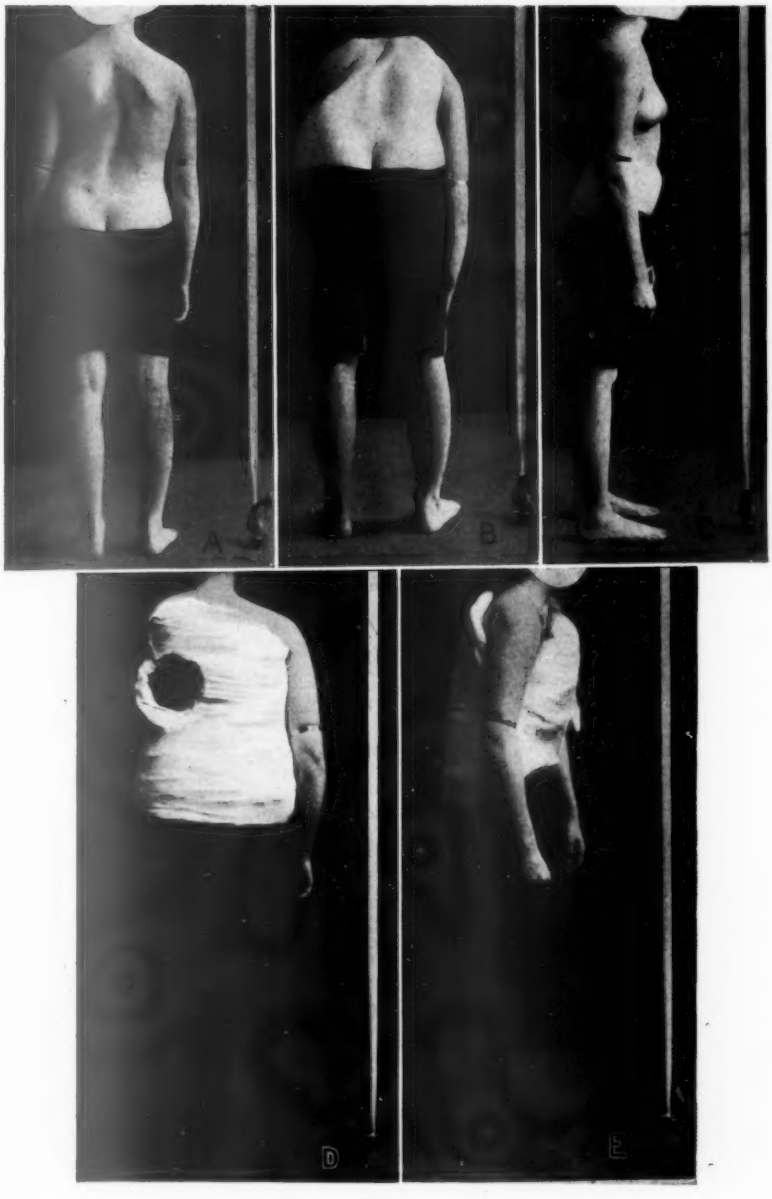


FIG. 1.

A, B, C.—Photographic record of case when first seen; right dorsal, left lumbar curve, with considerable dorsal rotation of the bodies of the vertebræ to the right, humping of ribs on the right side and flattening of left side of back (B) The white line is a plumb-line. C shows round back and lumbar lordosis. Note that weight of shoulders appears to be over the right foot.

D, E.—Appearance of "Abbott" jacket immediately after it was put on. Note (D) she is standing on her left leg instead of her right, and (E) she is standing, in the lateral view, in a position of total kyphosis.

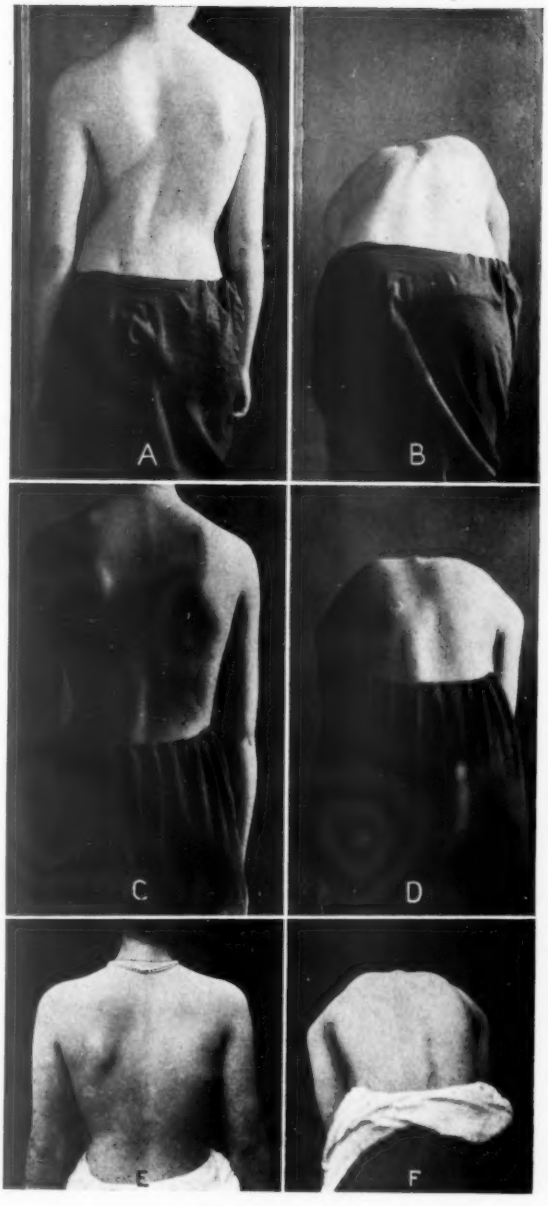


FIG. 2.

A, B.—Case when first seen in September, 1916.

C, D.—Condition in March, 1917, after two jackets. Ready for ordinary gymnastic treatment, which was carried out at home mainly by the governess, under periodic supervision by a trained gymnast.

E, F.—Photographs sent me by the patient in June, 1922, four years after she had last been seen by either gymnast or surgeon, showing voluntary maintenance of correction.

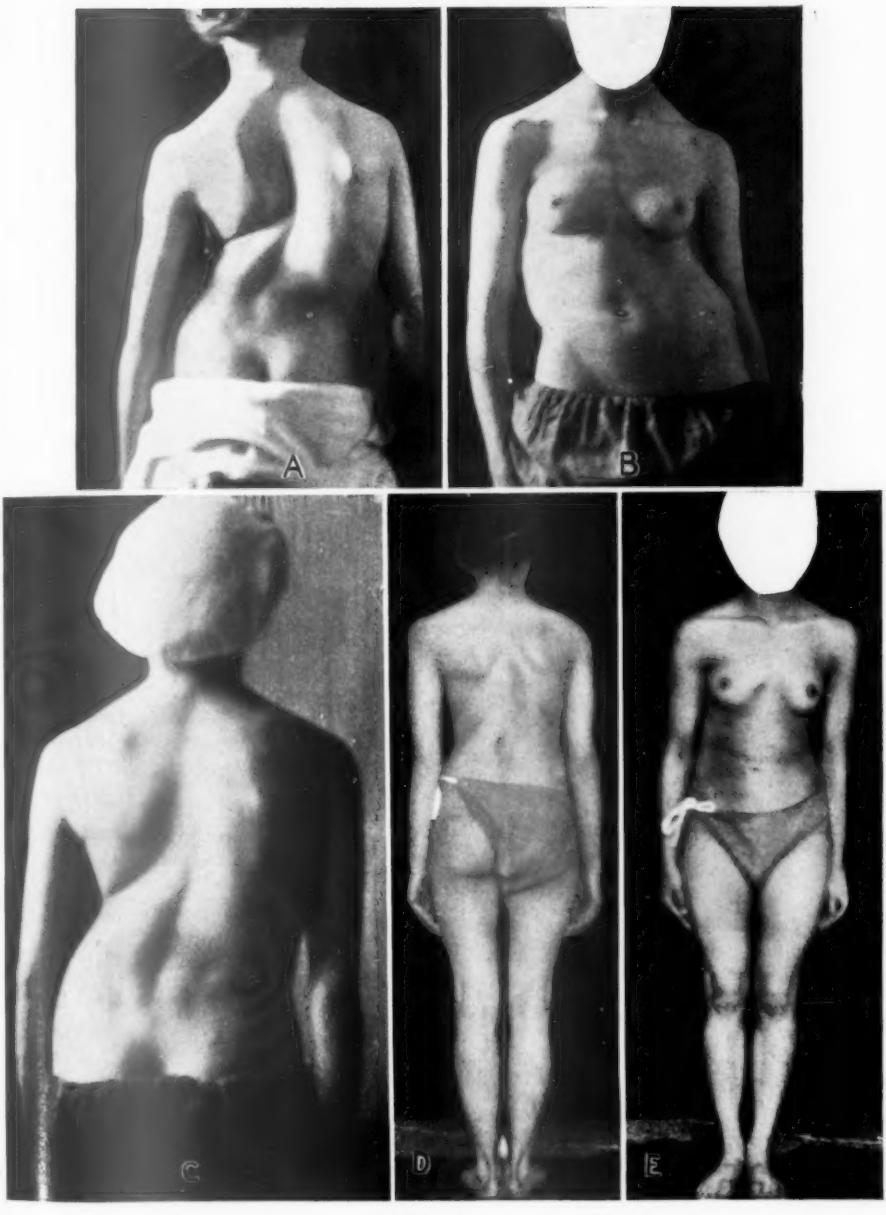


FIG. 3.

A, B.—Girl, aged 14, condition in August, 1924. History of fall when 7 years old, bad attack of "influenza" during the war. Also has had exophthalmic goitre.

C.—October, 1924, after first jacket.

D, E.—March, 1926, when patient was wearing short retentive jacket and continuing exercises.

This patient was seen on the moving pictures breathing freely with the side which was collapsed.

trunk, say congenital absence of ribs on one side, destructive disease of bone, or thickened rigid pleura after an empyema, is sure to breathe asymmetrically and also to stand asymmetrically. Conversely the child who, for any reason, stands habitually on one foot more than the other, will also expand the chest more on one side than the other, which may lead to adaptive changes in growth. Thus a school child may acquire a scoliosis from a transitory pleuro-pneumonia or a temporary injury to a foot, for the one induces unequal breathing and the other an unequal stance.

In order to treat scoliosis by splinting, it is necessary to discover first what the really essential deformities are, and secondly, how to apply splints so as to prevent increase of deformity and at the same time encourage free movement in the direction of correction in accordance with Sir Robert Jones's principles.

Taking for purposes of explanation what may be called a simple ordinary type of scoliosis, say with a curve convex and rotated to the right in the dorsal region, and convex and rotated to the left in the lumbar region. The writer now recognizes three essential factors in such cases. Any jacket or splint used to correct scoliosis must deal properly with each of these. (1) A scoliotic thorax expands during respiration on the humps, i.e., in our sample case the right side behind and the left side in front, while the flattened parts of the chest right-front and left-back hardly move. This was fully described by Michael Hoke in 1904, with the further pregnant observation that compression of the bulges immediately caused the movements of the flattened parts to increase. Hoke, therefore, applied plaster jackets to compress the long diagonal of the thorax, leaving windows for the flattened parts of the chest to expand. (2) In order voluntarily to breathe asymmetrically in this way it is necessary to stand unequally on the feet. The jacket must, therefore, push the patient's centre of gravity off the foot on which he usually stands most, on to the other foot. Thus reversing the stance, and with it the associated respiratory movement. (3) Full flexion of the lumbar part of the vertebral column: the writer was groping for this when Abbott, in 1911, published his first paper on flexed jackets with breathing windows. Up to 1911-12 the writer was trying to twist and correct scoliotics with the patient face down—a position which puts the back into one long lordosis. With the patient on the back a total kyphosis is easily obtained. This is essential to success, for as long ago as 1854 William Adams remarked that the lumbar part of the deformity of scoliosis is really an asymmetrical lordosis.

Those who are curious to follow this line of thought may consider an ordinary case of round hollow back in a child (kypho-scoliosis). This deformity is typical of the child who is out of condition and lacking in energy. The usual posture is (1) The toes turned out and the scaphoids dropped too near the ground (the position of pronated or "flat" foot). (2) Slack knees or slightly knocked knees. (3) Pelvis tilted forwards, i.e., hips flexed. The abdominal muscles are, therefore, relaxed and the abdominal viscera dropped. (4) The lumbar part of the vertebral column is in lordosis. (5) The dorsal part is in a compensatory kyphosis. (6) The child may also be a mouth breather. In this atonic slack position the diaphragm is practically useless, respiration is carried on by the round back, and most of the expansion of the ribs is posterior.

Now if such a child, instead of standing equally on the two feet, stands more on one foot than on the other, the kypho-lordosis becomes twisted, and we call the deformity "scoliosis." If the child stands more on the right foot, the pelvis instead of tilting straight forward, tilts forward and to the left, so that a slouching lumbar lordosis increases the left lumbar deviation, as pointed out by Adams. The dorsal kyphos must of course go to the right to maintain balance.

[The series of pictures appended[1] illustrate the results obtained as the writer gradually grasped the interdependence of the various elements of deformity, and learned to correct them by jackets and by breathing exercises, which associated posture with breathing. The moving pictures showed such patients in actual motion illustrating the interdependence of posture and respiration.]

1 From "The Robert Jones Birthday Volume," Oxford University Press, 1928.

[*December* 3, 1929.]

DISCUSSION ON MINOR INJURIES OF THE ELBOW-JOINT.

Mr. W. H. Ogilvie: A discussion upon minor injuries of the elbow-joint is not concerned with gross fractures or complete dislocations. It comprehends partial fractures of the bones composing the joint, partial dislocations, tears of the capsule, traumatic arthritis, and injuries of the muscles acting upon or overlying the joint. I propose, then, to enumerate the common traumatic affections of the elbow under these headings, in order to map out the ground upon which our discussion can proceed.

In order to place this classification on a reasoned basis, it is necessary to consider briefly the structure and mechanism of the elbow-joint, and to seek the answer to several simple questions: What are the movements of the elbow-joint; what muscles cause and what structures limit these movements; what are the defences of the joint and what its weak points; and what is the mechanism by which the injury is brought about.

The elbow-joint of surgery comprises the elbow-joint of anatomy, that is the humero-ulnar and radio-humeral joints, and also the superior radio-ulnar joint. The movements that occur at this joint are those of flexion and extension, pronation and supination.

The humerus articulates by the trochlea with the greater sigmoid cavity of the ulna, and by the capitellum with the cup-shaped hollow on the superior surface of the head of the radius. The greater sigmoid cavity of the ulna is divided by a transverse interruption, wider on the inner aspect, into an olecranon and a coronoid segment; the cartilage covering the coronoid segment is continuous with that lining the lesser sigmoid cavity. The head of the radius articulates by its superior aspect with the capitellum and by its rim with the lesser sigmoid cavity. The joint is enclosed in a complete capsule. The anterior and posterior ligaments are weak structures, composed of irregularly disposed fibres, which are continuous with the lateral ligaments and adherent to the overlying muscles. The internal lateral ligament is fanshaped, and three portions are described: an anterior band passing from the anterior aspect of the internal condyle to the inner margin of the coronoid process, a posterior band passing from the inferior-posterior part of the internal condyle to the inner margin of the olecranon, and a transverse band bridging the notch between the coronoid and the olecranon processes. The external lateral ligament is a strong, flattened band which is attached above to the lower and back parts of the external condyle, and below to the orbicular ligament, and through it to the ends of the lesser sigmoid cavity. The orbicular ligament is attached to the anterior and posterior margins of the lesser sigmoid cavity, forming four-fifths of an osseo-tendinous ring; it is not directly attached to the radius, but, being of funnel shape, the narrower distal circumference closes on its neck. The synovial membrane lines the capsule and clothes the intracapsular portions of the bones which are not covered with cartilage, notably the neck of the radius and the inner edge of the trochlea. It closes the articular cavity at the lower unattached border of the orbicular ligament.

Such are the main facts about the elbow, which can be found in any anatomical textbook. But anatomists appear to find this joint lacking in real fascination.

They refer to its movements as those of a hinge, and omit those dissertations upon function which lend life to their pages describing knee, hip and shoulder. Yet a detailed study of the elbow discloses facts of the greatest interest, which in turn suggest views concerning its function and movements, and throw light upon the mechanism of its injuries. Such a study has been undertaken by A. R. Thompson, the genito-urinary surgeon, to whose stimulating article in the *Journal of Anatomy* I have turned for instruction and guidance.

FIG. 1.—Lower end of right humerus from below.

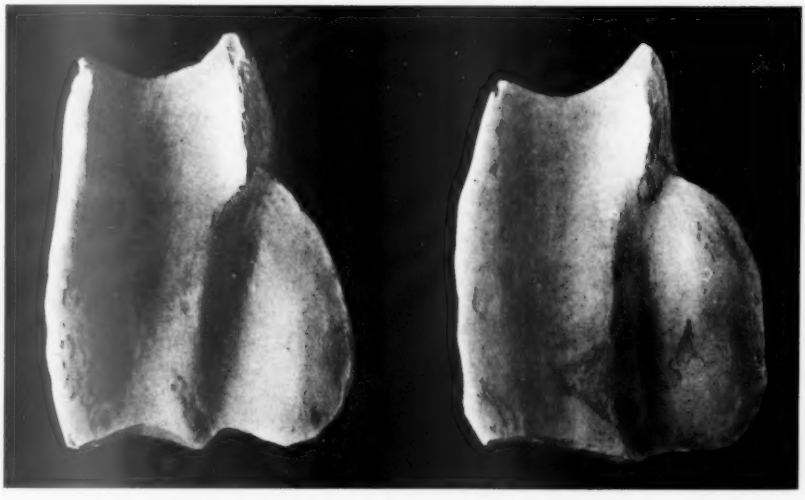


FIG. 2.—Projection moulds of lower end of right humerus.
(1) By rolling. (2) by unfolding.

The cubital articular surfaces of the humerus and ulna bear evidence of a division of the humero-ulnar joint into two parts. On the ulna the division is marked by the rough area crossing the sigmoid cavity transversely; on the humerus by a tubercle on the outer lip of the trochlea (fig. 1). In its posterior two-fifths the trochlea is a symmetrical groove, with sides of approximately equal slope, and bordered on each side by a prominent lip. The inner lip continues throughout the extent of the trochlea, and describes about three-quarters of the circumference of a circle. The outer lip ends suddenly at the junction of the posterior two-fifths and the anterior three-fifths of the trochlea, in the prominent tubercle

mentioned above. This tubercle is situated behind the inner part of the capitellum. In front of this point the capitellum, which belongs only to the anterior part of the articular surface, cuts away the outer lip of the trochlea and displaces about one half of the outer side of the trochlear groove. Put in another way: if the trochlea were continued forwards as it starts behind, it would cut off about one-third of the capitellum. The relation of these surfaces to each other can be demonstrated admirably in the method suggested by Thompson of forming what may be called a Mercator's projection of the articular end of the humerus. This may be done either by moulding wax round the lower end and unfolding the mould, or by rolling the lower end of the humerus round the transverse axis of the elbow-joint on a flat sheet of wax. In each case a cast taken from the wax mould will portray the various surfaces faithfully, except in the respect that they are now on a plane and not on a curve. I show casts obtained by each of these methods (fig. 2). They demonstrate very well the outer lip of the posterior part of the trochlea, ending in a prominent tubercle behind the capitellum, and suggest a division of the joint into a posterior trochlear portion and an anterior condylar portion, resembling in a general way the trochlear and condylar portions of the lower end of the femur.

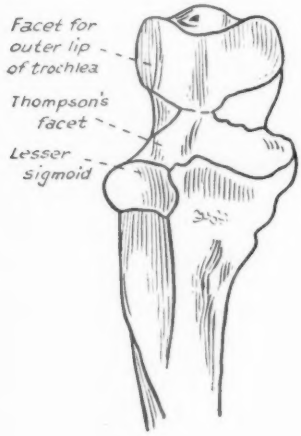


FIG. 3.

On the outer part of the olecranon surface of the ulna there is a facet of crescentic shape which, in full extension of the elbow, lies in contact with that part of the outer lip of the trochlea which ends at the tubercle; in flexion beyond a right angle this facet is entirely uncovered (fig. 3). There is another accessory facet on the coronoid part of the greater sigmoid cavity, which I have only found described in Thompson's paper. This lies between the outer part of the transverse groove dividing the greater sigmoid cavity, and the upper edge of the lesser sigmoid cavity, and is of triangular shape. It is uncovered in all positions of the joint except that of full extension, when it comes into contact with the tubercle on the trochlear lip.

Turning to the capsule of the elbow, we find that in the greater part of its extent it adds little to the strength of the joint. The posterior portion, especially, is extremely weak and intimately blended with the extensor muscle of the joint, in which respects it resembles the anterior part of the capsule of the knee. The anterior part is somewhat stronger. The strength of the joint lies in the lateral ligaments, or, to be more exact, in special parts of these: these defences of the elbow

are the anterior band of the internal lateral ligament and the posterior band of the external lateral ligament. The remainder of these ligaments, as described in text-books, are of dissecting interest, but no real strength; the portions alluded to, however, are very firm bands, which remain taut in all positions of the joint, and which maintain in the dissected specimen a perfectly stable articulation even if all the rest of the capsule is cut away. Their points of humeral attachment, the anterior border of the internal epicondyle and the most posterior part of the external epicondyle, therefore indicate the axis of movement of the humero-ulnar joint. In this way they resemble the lateral ligaments of the knee-joint.

Two points about the synovial membrane call for comment. The first is the sacciform nature of its cavities in both the upper and lower radio-ulnar joints, which clearly allow for an up-and-down as well as the more commonly recognized rotary movement between the two bones (fig. 4). The second is the high degree to which intracapsular synovial projections are developed in the elbow. The fatty pads in the olecranon, coronoid and supracapitellar fossæ are well known. They are the local expressions of a principle seen throughout the body, that wherever structures lying

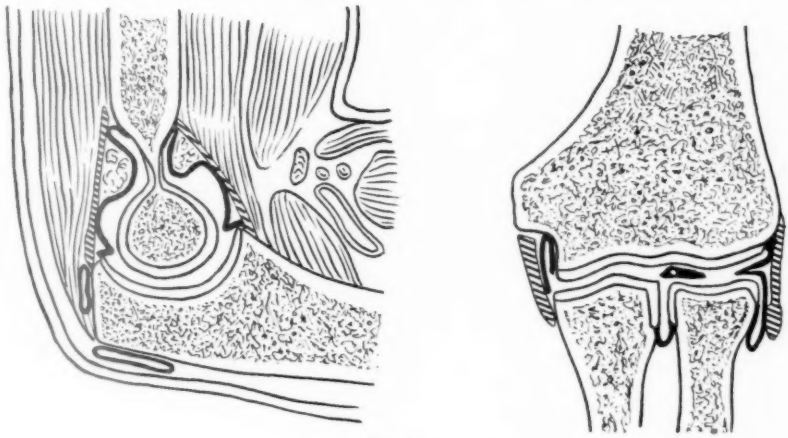


FIG. 4.

in rigid compartments are liable to undergo changes in position or size, a buffer of displaceable fat is introduced to allow for such changes. The mechanism in joints is therefore identical with that which allows the eye to move in the bony orbit, with the fat of the ischio-rectal fossa which permits passage of fœtus or fæces through the rigid pelvic outlet, and the crural canal, which can only be excused on the grounds that it enables the femoral vein to increase its sectional area under conditions of muscular activity. In addition to these large collections of fat which play the part of ballast, there are a series of synovial fringes, representing the mechanism whereby nature overcomes the problem of the sharp edge. In a hinge-joint, were two exactly fitting surfaces opposed, the periphery of the concave one would terminate in a knife edge, which would be liable to fracture from muscular action or direct violence: borders of concave surfaces are therefore rounded off, and the gap between this rounded edge and the capsule is filled by intra-articular pads of cartilage or synovial fringes. Such fringes constantly occur in the shoulder, hip, knee, and ankle, but are never referred to in the descriptions of textbooks. In the elbow they are found immediately adjacent to the attachment of the capsule to the

coronoid and olecranon processes. They reach their greatest development on the inner aspect of the orbicular ligament, where they form a crescentic meniscus having an arc of seven-eighths of a circle intervening between the peripheral parts of the radial head and the capitellum. The resemblance of this synovial disc when seen from above to one of the semilunar cartilages of the knee is very striking (see fig. 8).

A study of these points in the anatomy of the elbow-joint points to certain inevitable conclusions. Firstly that the elbow is primarily designed for use in positions between 110° and 170° of extension, and between half and three-quarter pronation. Within this range, and only within it, the olecranon portion of the greater sigmoid cavity is fully engaged by the trochlear surfaces of the humerus (fig. 5, i): in flexion beyond 110° it rides in a groove which is shallow on the outer side, so that part of its bearing surface is exposed (fig. 5, ii). The real humero-ulnar articulation is between the anterior or condylar parts of the trochlea and the coronoid articular

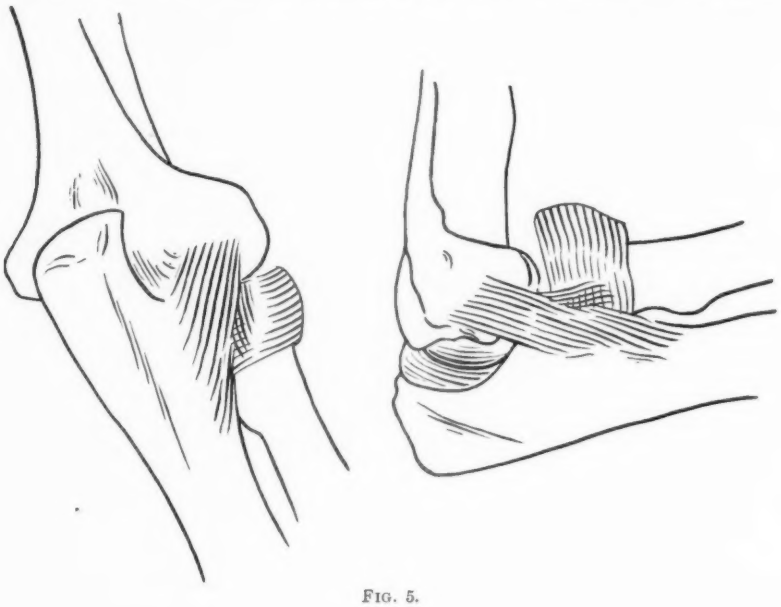


FIG. 5.

surface on the olecranon. The olecranon part of the ulna combines the function of a muscular process like the great trochanter of the femur, and a steadying and guiding mechanism comparable to the malleoli at the ankle. This latter function is only fully called into play in the later degrees of extension, when the olecranon sinks deeply into the trochlear portion of the humeral articular surface, fixing the ulna in the line of prolongation of the humerus much in the way that the tabernacle steadies a hinged mast when it is fully erect.

That the humero-ulnar articulation is the real joint between the arm and the forearm is shown by the breadth of the articular surfaces on the inner side of the joint compared to their slender character on the outer side, by the massive size of the upper end of the ulna compared to that of the radius, and by the absence of any real disability or loss of strength after excision of the head of the radius. The ulnæ form the platform upon which the radius, carrying the hand, is free to rotate.

The normal relation of the radius to the ulna is one of half to three-quarters pronation. This is shown on the bones by the articular surface on the circumferential part of the radial head, which, immediately above the bicipital tuberosity attains a breadth of some 12 mm., from which it rapidly diminishes in either direction to a depth of 2 mm. over that half of the circumference which lies opposite to the tuberosity. This broad surface, in the position of half pronation, corresponds accurately to that of the lesser sigmoid cavity of the ulna, which is also deepest in the middle and narrows in front and behind that point. In other positions, co-aptation is incomplete. Thompson explains this broad area on the radius by suggesting that it supports the tendon of the biceps in the position of full extension and supination, and compares it to the similar facet on the front of the head of the femur which supports the psoas tendon. That this explanation is not the likely one is shown by reference to the articular surface of the lower end of the ulna which has a similar disparity in depth, the deep portion being fully engaged by the sigmoid cavity of the radius in the same arc of pronation. Moreover, the position of full supination and full extension, in which the tendon of the biceps lies on the radial head, is most exceptional in the elbow, whereas the psoas tendon presses upon the femoral head in the habitual position of the hip.

This position of half to three-quarter pronation is, if we consider it, that in which the forearm is held in all the common uses of the arm: moreover, it is the constant use of this position that provides the only acceptable explanation of the "carrying angle" at the elbow.

The carrying angle is one of 10° to 15° of abduction in the extended ulna from the line of the shaft of the humerus, and is present at all ages, and in the full-time fœtus. The statement that the ulna is carried inwards in full flexion is not supported by an examination of the skeleton. The angle diminishes as the arm is flexed, and in the fully flexed position it disappears, the two bones lying in the same plane. This abduction of the ulna in full extension is due to the varying depth of the inner lip of the trochlea, which is deeper than the outer lip below, but equal to it in its upper part. The common explanation of the purpose of the carrying angle is that it is a provision of nature which allows man, or rather woman, to carry pails of water without bumping the knees at each step. Apart from the obvious objection on evolutionary grounds that elbows were invented millions of years before pails, it must be insisted again that the position of full extension and full supination in which the carrying angle becomes obvious, is a wholly artificial one. To display one's own carrying angle, one must exert a degree of muscular force which is quite unpleasant: to demonstrate it in a patient, one must assist his voluntary efforts by forced passive outward rotation of the hand. Anybody who tries to carry a two-gallon petrol tin for one hundred yards in a position of full supination will quickly realize that this is not even the attitude intended by nature for carrying buckets. Put the forearm, however, in its position of use—that of nearly complete extension and three-quarter pronation—and the purpose of the carrying angle becomes apparent, which is to allow the radius to lie in the long axis of the humerus. When the boxer delivers his blows, arm and forearm are in one straight line because of the carrying angle. Were the ulna a continuation of the line of the humerus, such a blow with the extended and pronated arm would tend to spring the elbow inwards.

The ready position, or position of rest, of the elbow is one of slight flexion and rather more than half pronation. There is one muscle, the brachio-radialis, whose main purpose appears to be to sling the elbow in this position, and to flex it in this position during action. Supination and pronation from this point are consciously active movements, and the forearm always tends to return to mid-pronation. From this position it is moved to carry out various functions. It follows from the high specialization of the human hand that the uses to which it is put fall into two distinct categories—those which concern the outside world and those which concern

the individual. The elbow then, which governs the position of the hand, is called upon for much more complex movements than the knee, whose final outpost is only concerned with the ground. The ranges within which the elbow-joint is called upon to act may be called, borrowing the term from the physiology of the central nervous system, the exteroceptive range and the proprioceptive range. The exteroceptive range embraces the positions from about 90° to 170° of extension and from half to full pronation. In this range the arm is used for attack, for cutting or rending food, for climbing, for supporting the body, and for handling tools or weapons. It is, therefore, within this range of movement that stresses are likely to fall on the elbow-joint and damage to occur. In this range the articular surfaces on the elbow are fully engaged and the joint is a stable one. The proprioceptive range, from a right angle to full flexion and from half to full supination, embraces all those movements

FIG. 6.

concerned with the person, which have risen from the simple wants of primitive man to convey food and remove parasites, to the very complex movements of the civilized toilet. In this range the articular surfaces are poorly adapted and the joint depends to a great extent on ligaments for its stability, but it is seldom exposed to injury. Full supination is rarely used in extension or full pronation in flexion.

Full extension at the elbow does not occur in natural use, the elbow being rarely extended beyond 170°. Further extension requires a considerable exertion of force, and the position cannot be maintained for long. In a position of full extension the tip of the olecranon comes in contact with the floor of the olecranon fossa, and the tubercle on the outer lip of the trochlea presses upon the triangular facet on the ulna described by Thompson. That this position is not a normal one in use is suggested by the observation that the head of the radius is then only in contact with the capitellum in the anterior half of its upper surface (fig. 6). Hyper-

extension rarely occurs. The apparent hyper-extension seen in some women is due to the demonstration by extreme supination of an exaggerated carrying angle. That up-and-down movements of the radius on the ulna may occur is suggested by the sacciform nature of the synovial cavities in the upper and lower radio-ulnar joints. That they do occur can easily be demonstrated under X-rays. In the position of rest there appears to be a slight space between the head of the radius and the capitellum (fig. 7, i). On making firm pressure on the radially abducted hand, the radius can be pushed slightly up, probably displacing the synovial fringes (fig. 7, ii). On pulling the hand into ulnar adduction, the radius moves an appreciable distance away from the humerus (fig. 7, iii). Upward movement of the radius on the ulna appears to be checked by the interosseous membrane, downward movement by the oblique cord. What purpose these movements serve is difficult to conjecture, but they invite comparison between the outer side of the elbow and its homologue in the lower limb, the inner side of the knee (fig. 8). In each case we have a broad lateral ligament whose lower attachment passes well down the shaft of the distal bone compared with the cord-like one on the opposite side, an inter-articular disc

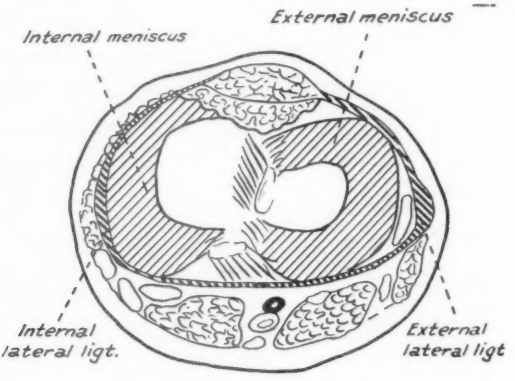
i. ii. iii.

FIG. 7.—Radiograms of right elbow: (i) at rest; (ii) when pressure is applied to the radially adducted hand; (iii) when the hand is pulled into ulnar abduction. (Radiograms by Dr. H. M. Worth.)

intimately attached to the ligament, a certain amount of gliding as well as hinge movement, the possibility of abduction to a limited degree, and a broad covering of muscles checking this abduction.

The majority of injuries, major and minor, which are seen in the elbow-joint are due to indirect violence, the action of a force transmitted along the forearm to the elbow. In addition, the elbow region is exposed to direct violence, either by falls, or by blows upon the arm raised in self-defence. The trochlea, and still more the capitellum, are carried well forwards upon the lower end of the humerus, so that a force transmitted directly upwards along the bones of the forearm would tend to fracture these portions of the articular end forwards into the joint. In the fully extended position also, the posterior lip of the capitellum lies like a ridge transversely across the radial head, and would tend in compression to split it across its diameter. This position of full extension is, however, rarely assumed, and if assumed cannot be maintained in face of a force transmitted along the arm. Turner Thomas has demonstrated, by experiments on the cadaver, that the force of a fall on the outstretched hand tends to flex and not to extend the elbow. He made this observation

when attempting to reproduce in the cadaver, fractures of the head of the radius, by holding the arm in an extended position and delivering blows on the head of the humerus with a heavy mallet. Such blows, as I have confirmed to my own satisfaction and occasional pain, always tend to flex the arm at the elbow, never to hyper-extend it. Indeed, hyper-extension at the elbow could only occur as the



RIGHT KNEE FROM ABOVE: SECTION.

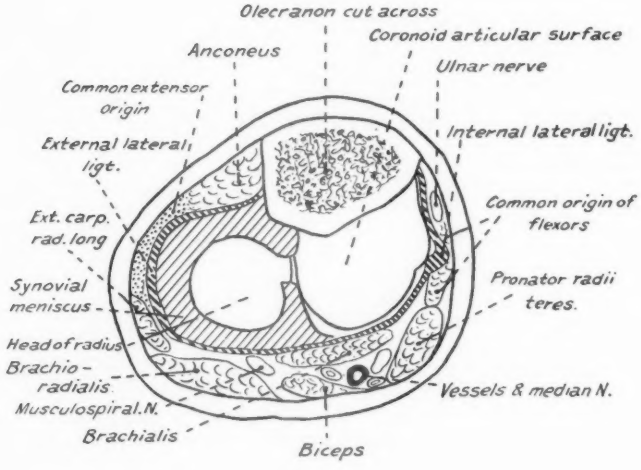


FIG. 8.

result of a fall on the hand when the wrist was fully dorsiflexed and the arm fully supinated, so that the line of transmitted force passed behind the centre of rotation of the elbow. Such a position cannot happen. In the ordinary fall on the outstretched hand, when the palm strikes the ground, the line of force coming downwards through the humerus meets the line of resistance coming upwards from

the ground, chiefly through the radius (fig. 9). Both of these lines pass out of the limb posteriorly at an angle to each other. When the forces are sufficient to break the skeleton at or near the joint, the remaining momentum drives the two fragments in the lines of these forces. The usual result is either the typical supracondylar

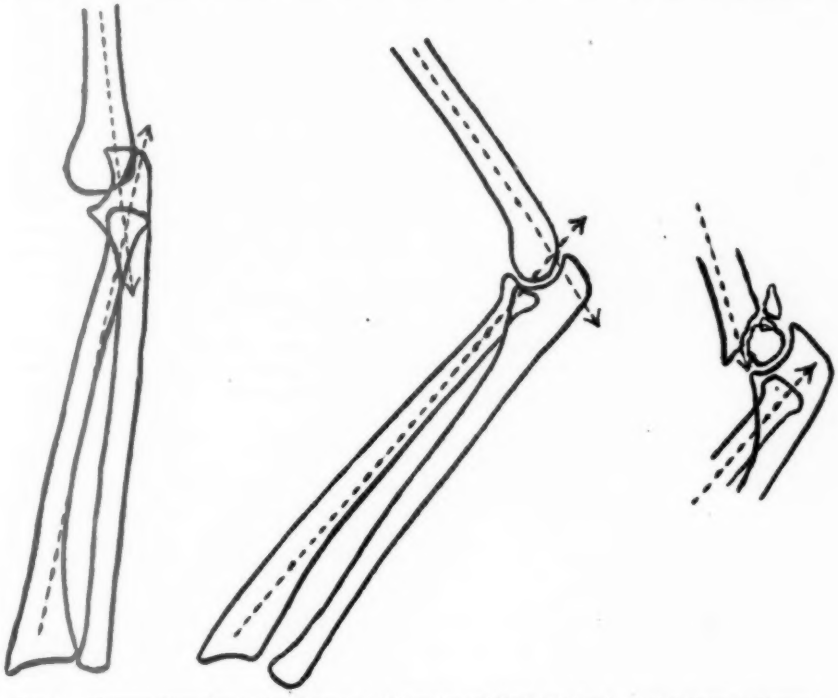


FIG. 9.—Stresses occurring at the elbow in a fall on the outstretched hand. (After Turner Thomas.)

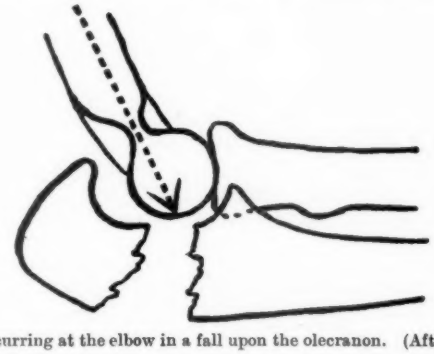


FIG. 10.—Stress occurring at the elbow in a fall upon the olecranon. (After Turner Thomas.)

fracture of the humerus, or the typical backward dislocation at the elbow. The involvement of the radius in fractures due to falls on the outstretched hands depends largely on the degree to which force applied to the radius is passed on to the ulna. The interosseous ligament is to some extent relaxed in full pronation:

the radius in full radial adduction of the hand is forced against the capitellum. Therefore falls upon the hand in a fully pronated and radially adducted position, such as occurs when the arm is thrown well forwards of the body, would tend to cause a fracture of the head of the radius, while if the hand were close to the side it would tend to strike the ground in a position of partial supination and ulnar adduction, and a dislocation of the elbow or fracture of the humerus would be more likely to result. If the arm is partly flexed at the time of impact, further flexion occurs so rapidly that the main force of the blow is taken on the most exposed part of the elbow, the olecranon. The body weight travelling down the humerus then drives the trochlea like a cold chisel into the sigmoid cavity of the ulna, and, if the force is sufficient, the typical transverse fracture of the olecranon process is the result (fig. 10). Further application of the same force carries the coronoid part of the articulation, and with it the radius, upwards in front of the humerus. Direct violence usually implicates the ulna, and may produce the simple transverse or comminuted fractures of this process. However, the violence, according to the size of the striking object and the point of impact, may cause a fracture of any part of any of the constituent bones. Forward dislocations of the radius may thus be produced by the impact of a rounded body upon the radial head.

Classification of Minor Injuries.—The most simple classification is an anatomical one based upon the structure mainly involved. One common injury, tennis elbow, escapes any such scheme, since surgery has given it a name, but so far no local habitation.

Minor Injuries of the Elbow.

(I) *Minor Fractures.*—(1) *Humerus.*—(i) Supracondylar fractures without displacement, (*a*) cancellous crush fractures, (*b*) fissures. (ii) Fractures of internal epicondyle. (iii) Fractures of internal condyle. (iv) Fractures of trochlea. (v) Fractures of external epicondyle. (vi) Fractures of external condyle. (vii) Fracture of capitellum. (2) *Ulna.*—(i) Partial fractures of the olecranon. (ii) Fractures of the coronoid process. (3) *Radius.*—(i) Fractures of head. (ii) Fractures of the neck without displacement. (iii) Fractures of the tubercle.

(II) *Minor dislocations* of radial head.—(i) Subluxation forwards. (ii) Pulled elbow.

(III) *Sprains of the Joint Capsule.* (V) *Traumatic Enlargement of Bursæ.*

(IV) *Injuries of Muscle.* (VI) *Tennis Elbow.*

Supracondylar fractures without displacement are common in childhood, and may easily be missed in a poor radiogram: a stereoscopic examination is often necessary to demonstrate them satisfactorily. Bruises or sprains of the elbow in a child give rise to little limitation of movement, and only a small amount of swelling. If there is much bruising round the lower end of the humerus and movements are considerably restricted, a fracture is almost certain, even though all the bony points are in their normal relation. Such fractures, if there is no displacement of the articular surfaces, demand only a few days of immobilization, and the essential movements of flexion and supination should be actively exercised from the start. In the absence of periosteal stripping there is no risk in early movement of stimulating bone formation.

Isolated fractures of the internal epicondyle are distinct from fractures of the internal condyle. They are due as a rule to muscular violence, are usually seen in adults, and the joint is not involved. The loose fragment is pulled downwards by the common origin of the flexor group. Even when treatment in full flexion has been adopted, correct position and bony union are seldom obtained, but the resulting disability is slight.

Fractures of the internal condyle are oblique, and enter the joint anywhere between the inner edge of the trochlea and the capitellum. They are commonly seen in children, and are usually due to falls on the outstretched hand. The broken

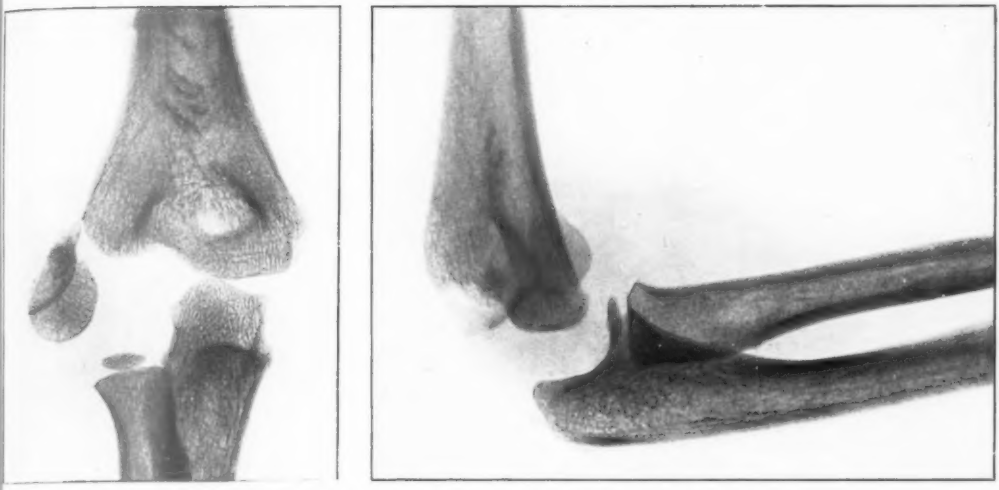


FIG. 11.—Fracture of capitellum. (Radiograms by Dr. H. Courtney Gage.)

FIG. 12.—Fracture of capitellum. Same case as Fig. 11, one year after operation. (Radiogram by Dr. H. Courtney Gage.)

fragment tends to ride upwards. This displacement is easily reduced by traction, but recurs when traction is released. Fractures of the trochlea alone are very rare.

The external epicondyle is such a small process that only cracks due to direct injury, or detachments of flakes of bone caused by muscular violence or by indirect force applied through the capsule, can be included as isolated injuries. Fractures of the external condyle are much commoner, and are due to force transmitted from the ground through the radius to the capitellum. The condylar fragment is displaced upwards so that cubitus valgus is a common sequel in cases that have not been treated efficiently. The frequent occurrence of late ulnar palsy as a sequel of fractures of the external condyle has been fully discussed by Platt.

Isolated fractures of the capitellum are rare. The injury is mainly intra-articular, so that there is no deformity of the limb and the bony points round the elbow appear normal. Excessive limitation of function and swelling of the joint capsule with blood alone indicate that the injury is more than a sprain.

I have had three cases of capitellum fracture, two within a few days of each other in 1928. In both of these the capitellum was completely detached, lying free in the joint in front of the lower end of the humerus and rotated on its axis. Any treatment apart from open operation was obviously futile.

The first case only came to hospital seven weeks after the injury. I was unable to replace the fragment in a satisfactory manner and therefore excised it. The result a year later is full pronation and supination and flexion from 135° to 90°.

The second patient was a child aged 6, who was seen immediately after the injury (fig. 11). The joint was opened by a lateral incision: the fragment, which was entirely loose, was rotated into position and the arm put up in full flexion. In this position there was no great tendency for the capitellum to become redisplaced, and there did not appear to be any need for direct fixation of the fragment. A year later there is full movement, with the exception of slight limitation in extension. Radiograms show that the capitellum has survived and remained in place, but that there are several loose bodies in the joint (fig. 12).

The force causing an isolated fracture of the capitellum must almost certainly be one from below, travelling directly upwards in the line of the humerus, either an external force applied to the capitellum with the elbow in flexion, or one transmitted along the radius with the arm in complete extension. If any flexion were present, a supracondylar fracture of the humerus, or one of the external condyle extending into the shaft, would be more likely. Direct violence is the more probable cause, for in indirect violence the head of the radius would almost certainly fracture before the more massive capitellum. In my first case the patient fell to the ground upon the flexed elbow; in the second the nature of the violence could not be ascertained.

In general I am averse to any method of intra-articular fixation in the elbow. Where loose fragments cannot be kept in place after reposition by posture, or by extra-articular wires or screws, I would advocate traction in the flexed position by a pin through the olecranon.

Partial fractures of the olecranon may take the form either of detachment of flakes of bone from the extreme tip by muscular violence, or of fissured fractures of the process without displacement, caused by falls on the elbow. Fractures of the coronoid process are occasionally caused by muscular violence, the pull of the brachialis tearing away a flake of bone: much more commonly they are the result of a posterior dislocation of the elbow.

Fractures of the radial head may be caused by blows on the elbow, but much more commonly they are the result of falls on the outstretched hand, the head being splintered against the capitellum. It is convenient to divide these fractures into three categories :—

(*a*) Those with loose fragments in the joint. (*b*) Those with gross deformation of the head, but no loose fragments. (*c*) Fissured fractures without much displacement.

The first type demands immediate operation in every case—arthrotomy and removal of the loose fragments and the remainder of the radial head. The second class requires removal of the radial head in the great majority. There is no method whereby the fragments can be moulded, or the faulty position corrected. If, therefore, the head is tilted as a whole out of the plane of its rotation, or if its shape after consolidation will be such that it cannot rotate freely, operation should be carried out as soon as possible.

The most common fracture is that in which the fragments retain approximately their normal position. We have only one means of affecting the fractured site, the moulding influence of the orbicular ligament on the fresh callus. I therefore believe that these fractures should be treated as sprains, and voluntary movements of rotation instituted from the beginning. If pain is at all marked after three weeks I excise the head, but I rarely operate for limitation of movement, for in my experience the range of rotation obtained after excision of the radial head is from 50% to 70% of the normal, while when early movements are employed a full range is often obtained.

Fractures of the neck of the radius are usually due to indirect violence. If the axis of the head is normal, they are treated for ten days in a position of flexion and full supination, after which movements are started. If the head is out of alignment it should be excised. Fractures of the tubercle are rare, and the result of muscular violence.

The minor dislocations which are possible at the elbow affect the radial head. Forward dislocation of the head of the radius is usually associated with fracture of the upper part of the shaft of the ulna, but may occur as an isolated injury, in which case it is usually the result of the head of the radius striking a rounded object on the ground in a fall. Partial subluxations are not uncommon, and are difficult to demonstrate in any but stereoscopic radiograms. Pulled elbow, or partial subluxation downwards of the radial head, is one of the common accidents of infancy.

"Sprain of ligaments" is a clinical diagnosis and not a clear-cut lesion. Wrenching strains applied to the elbow may lead to tears of part of the capsule, or to detachment of ligaments from bone. Such injuries can only be inferred from the persistence of disability after an accident, and from the discovery of points of tenderness. Owing to the intimate association of muscular origins with the capsule, it is probable that muscles are nearly always involved as well as ligaments in these sprains.

Partial tears of any of the muscles round the elbow may follow any sudden unguarded movement. Such tears usually involve the muscle near its point of attachment, and affect the triceps, brachialis, or biceps. They can only be distinguished from the partial fractures, which are often caused by the same violence, by a negative skiagram and by the absence of crepitus. In individuals of pronounced muscular development ossification in the tendons of these muscles often occurs as age advances, and the fracture of such bony spurs by a sudden contraction may be the cause of acute and localized pain (fig. 13).

Traumatic myositis may follow prolonged and unaccustomed use of any muscle. This affection is common in the tibialis anticus after prolonged walks undertaken by the sedentary. I have seen it in the supinator brevis in a patient who had indulged in a spasm of carpentry. The whole muscle is tender to pressure, and, on passive movements, a soft crepitus may be felt over its site.

Bursæ, like any other structures, may be injured. Enlargement of the olecranon bursæ is too obvious to merit consideration by a scientific body, while traumatic affections of the other bursæ round the elbow are as a rule part of a more general injury and only diagnosed by guesswork.

Tennis Elbow.—Tennis elbow is one of the commonest disabilities of the elbow, yet it eludes classification, for its seat has been variously ascribed as muscular, bursal, periosteal, ligamentous and synovial.

The clinical features of tennis elbow alone are well recognized. It is found in the followers of those trades and devotees of those sports in which the arm is repeatedly jerked into full extension while the hand grasps some large instrument in a whole hand grip. In addition to tennis players, fly fishers, golfers, sabre fencers and workers with the hammer are afflicted. The onset is rarely sudden. During the course of a game the patient notices an aching in the outer side of the elbow, which is most marked in certain movements, unnoticed in others. After the game pain ceases, to reappear whenever the same action is resumed. The pain tends to get progressively worse with use, till, in the most severe cases, a feeling of soreness is constantly present, and an aching on exertion which leads to abandonment of the game. With long abstinence the pain may disappear, but is very apt to return

FIG. 13.—Fracture of spur in brachialis tendon. (Radiogram by Dr. H. Courtney Gage.)

when the same action is resumed. When the injury is established, an additional symptom is found in many cases, a momentary weakness, or rather inhibition, which occurs chiefly on lifting small objects, such as a teacup, so that the object is dropped. The inhibition is entirely out of the patient's control.

On examination a definite localized tender spot is found in all cases on the outer side of the joint in the neighbourhood of the external epicondyle. Clenching the hand is painful. Apart from these two points no abnormality can be discovered. There is, in most cases, no swelling or other alteration in the contour of the joint and X-ray examination is negative. Passive movements are painless in all directions.

The main theories which have been advanced to explain tennis elbows are: (1) The tearing of some fibres of muscles arising in the neighbourhood of the external epicondyle. (2) Periostitis of the condyle or epicondylar ridge, either traumatic or infective. (3) Tears of the capsule involving either the external lateral or the

orbicular ligament. (4) Arthritis of the radio-humeral joint. (5) Inflammation of the radio-humeral bursa (Osgood).

Osgood's paper, stimulated by a personal experience of tennis elbow, and worked out with careful clinical, anatomical, and pathological reasoning, accompanied by reports of successful operations upon two cases in addition to his own, is one of the classics of orthopædic surgery. Osgood's bursa lies immediately distal to the external epicondyle, deep to the common origin of the extensor group, and between this and the radio-humeral joint. In his case this region was exposed under local anæsthesia, and a mass of bluish grey tissue was found, but no bursal cavity ; this tissue was excised and found to be connective tissue and fat, with some evidence of inflammation. The symptoms were permanently relieved as the result of this operation.

I have attempted to confirm Osgood's observations by dissection and by inquiry among surgical friends, but have been unable to do so. In eight subjects examined, I could find no tissue underlying the common extensor origin beyond that fine areolar tissue which is the lubricant of all muscular planes. Nor have I heard of any surgeon in this country who has confirmed Osgood's observations in cases of tennis elbow, or obtained success by his operation. That such a bursa may occur cannot be doubted, but it cannot be called a normal structure—that is, one found in the great majority of individuals. All bursæ are in a sense abnormal, for they are absent at birth and arise as degenerations ; they may be formed almost anywhere in response to excessive movement or pressure.

From my own experience, I believe that the common clinical type of tennis elbow is due to the tearing of muscle fibres at their origin from bone. The point of maximum tenderness in my cases has been just above the external epicondyle on the lowest part of the external supracondylar ridge, rather than in front of the epicondyle. This part corresponds to the origin of the extensor carpi radialis longior. The radial extensors come into powerful action as fixers of the wrist-joint in those gripping movements which are known to cause the pain in tennis elbow, and to be employed in those games which cause it. On the other hand the brachio-radialis, which has sometimes been named as the muscle involved, is relaxed in forehand shots which will involve elbow extension. The probable lesion is a tearing of muscle fibres where they rise from the bone, with, in some cases, separation of some of the periosteum. The nature of the injury must be conjured up, for upon this, and upon the stage of its evolution, will depend the treatment which is most likely to afford relief.

Such minor injuries pass through three stages. When first inflicted, the stage of a recent injury, there is an effusion of blood and tissue fluids, local hyperæmia and round-celled infiltration. This stage is marked by continuous pain and diffuse tenderness. It is treated by firm pressure, massage and rest.

The second stage is one of early repair. The damaged structures are being replaced by vascular repair tissue, and the torn periosteum is laying down new bone. At this stage we find pain on certain movements only, an occasional ache, especially on changes of weather or temperature, and a localized tender spot. Here firm pressure, combined with movements, is best calculated to hasten the absorption of inflammatory products.

The third stage is one of permanent repair, when the granulation tissue has become avascular fibrous tissue. This stage is only productive of symptoms if the repair has been such as to limit movements, that is to cause adhesions. Avascular connective tissue cannot be absorbed, but it can be snapped. This stage then responds to manipulation.

But while these three phases are typical of a lesion that is purely traumatic, and of any injury in childhood, few traumata, in this age of conservative dentistry, run a purely sterile course. In most of the muscular strains, ligamentous tears and articular injuries that we meet in adults, the element of sub-infection, probably due to a streptococcus, is added. The effect of the lodgment of infection in an area of

repair is to prolong the second stage, that of vascular repair and of localized tenderness. Symptoms may continue almost indefinitely while the focus of sepsis is present, and the final stage is less likely to be one of complete resolution and more likely one of adhesion.

The stage of recent injury is rarely seen in tennis elbow, since the complaint is troublesome rather than disabling, and patients go on for some time with their game in the hope that the pain will wear off, before consulting their doctor. Cases are more commonly seen in the second or third stage, and it is upon our estimate of the pathological process that treatment must be based. If there is an active inflammatory reaction and vascular repair tissue, forcible manipulation will only lead to tearing and further reaction and will do more harm than good. If, on the other hand, tough avascular fibrous tissue is present, no amount of pressure, massage or movement will do the slightest good, while manipulation will effect an instant cure. Pain or soreness apart from movement, marked or diffuse local swelling or elasticity, suggest an active lesion. Strict limitation of tenderness, restriction of movement and the limitation of pain to periods of activity and to certain definite actions, all suggest adhesions. The first type of case I have treated most successfully by applying a small pad, either of felt or of wool smeared with iodex, over the tender spot, and strapping the elbow in supination at an angle of about 135°, to limit full pronation and full extension. The second type is cured, in the majority of cases, by the manipulative manœuvre described by Mills. The wrist and fingers are put into full flexion, the forearm into full pronation, and the elbow is then forcibly extended with the one hand, while the thumb of the other is pressed firmly on the external epicondyle. This manœuvre stretches the extensors of the wrist to their fullest extent. If the stage of the lesion has been correctly estimated a sharp crack is either felt or heard and cure is assured: no click, in my experience, means no cure. In all cases the possibility of an infective element in the pain must be considered, and the teeth, tonsils, and colon investigated as potential sources.

But tennis elbow is not as simple as this, or it would not have produced the abundant and conflicting literature which has accumulated around it. There are other types, and there are symptoms which cannot be explained on the simple basis of a tear at the origin of the extensor carpi radialis longior. I refer, in particular, to the sudden darts of sharp pain that occur in some cases, and to the momentary giving way that makes the patient drop whatever he is holding. This appears to be a true inhibition of movement and not merely an arrest of a movement that is painful. It is exactly comparable to that sudden giving way of the knee which makes the patient fall to the ground even though no great pain has occurred. This symptom in the knee is only seen with internal derangements, never with injuries of the capsule or surrounding muscles, and appears to be a protective inhibition due to the sudden stimulation of the nerves of joint sensation in the synovial membrane. It is not seen in locking caused by loose bodies which have no nerve-supply.

I believe then that some types of tennis elbow are internal derangements, similar in their origin and cause to injuries of the internal semilunar cartilage in the knee. I have already suggested a comparison between the inner side of the knee and outer side of the elbow, and have shown that movements of ulnar adduction can take place, separating the radio-humeral surfaces, similar to those which occur at the knee and lead to injury to the internal meniscus. Such movements, opening out the radio-humeral interval, are inevitable in the quick flicking movements of tennis or fly fishing, and in them the radio-humeral synovial fringes may easily get nipped. Such nipping, as in the knee, would be specially prone to come about if the fringes were already thickened by arthritis, and it is well known that a purely traumatic arthritis may be induced in the elbow by over use, particularly by the return to a strenuous game at the beginning of the season after a period of inactivity. Kreuscher has described such a condition in baseball pitcher's elbow; a similar mild arthritis is often seen in the out-patient department of a hospital—in labourers.

If then such a type of tennis elbow can be verified, its treatment is by arthrotomy and removal of the synovial fringes which are the cause of the inhibition. I have seen such cases, but since the conception of an internal derangement has occurred to me as their true explanation I have not come across one in whom the crucial test could be applied. I am not convinced that Osgood's own case was not of this nature. In my dissections to confirm the existence of the radio-humeral bursa, I have found it almost impossible to reflect the conjoined origin of the extensors from the capsule of the elbow without opening the radio-humeral joint. In his case this joint was opened, and it is possible that the relief attained was as much due to the relief of capsular pressure on a thickened fringe and the provision of a path for lymphatic drainage, as to the removal of some rather indefinite tissue. The point of tenderness he describes exactly overlies these radio-humeral synovial fringes. At any rate I intend upon the next occasion to explore the joint, and meanwhile put forward these views for your consideration.

In summing up, I should like to insist once again upon the two uses of the elbow—the exteroceptive, and the proprioceptive. The exteroceptive uses are the essential ones, and the muscles carrying them out are predominant. Therefore in minor injuries, when we expect and hope to regain full movement, we must concentrate on the preservation of the proprioceptive range, full flexion and full supination, which is most easily lost. When we expect movement to be lost, we must preserve the exteroceptive range, and allow the elbow to ankylose at an open angle and in pronation. For all trades, games, and occupations, and even for cutting up food this position is essential. The classical position for ankylosis which still holds its place in the textbooks, an acute angle with full supination, is only useful to those who cannot tidy their hair with one brush, or who feel the necessity to get both their hands to their mouth at the same time.

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Mr. Jenner Verrall said that internal derangements of the elbow-joint as a cause of tennis elbow had appealed to him for a long time, but he had not had the opportunity of proving whether or not he was right. He agreed with what Mr. Ogilvie said about Osgood's bursa. In his opinion the undoubted good effect of seeking for that bursa had been associated with the division of the very tense and taut fibrous tissue at the origin of the extensors. He regarded tennis elbow in some cases as a muscular rupture, but in the majority as due to a combination of circulating toxins and local trauma. Beneath the tense fascia on the outer side of the elbow-joint there was very little space between that and the head of the radius, and in the two or three cases of tennis elbow he had operated upon, seeking for the bursa, there had been a very much thickened and dense adhesion between the tense fascia and the very near head of the radius. The combination of the opening up of that, combined with a treatment based on the circulating toxin, caused the cure which followed that very simple operation. Much the same explanation applied to the cases which were cured by Mills' manipulation. In his own experience this method of manipulation was successful in quite one-third of the cases; there occurred a rupture of the adhesions which could be heard some distance away. It could be explained equally well by an internal as by an external derangement.

Strains on the internal side of the elbow, such as were met with in so-called "golf elbow," were practically all due to over-action and strain of the flexor carpi ulnaris; they were due to forcible abduction of the wrist, causing tension on this muscle. This, carried further, would lead to that displacement of the ulnar nerve and chronic neuritis which these patients complained of, and which Mr. Harry Platt so well described, and which could only be cured by displacement of the nerve to the front of the elbow.

He considered dislocation of the head of the radius forward a serious matter, because, although it did not limit flexion of the elbow, it caused a musculo-spiral neuritis. In children it was important to get the head of the radius back quickly. In a child who had had the head of the radius displaced forwards for three months he had been unable to replace the head, even by open operation. If the head would not stay in place he advocated the construction of a new orbicular ligament from the fascia lata, an operation which he had done successfully several times.

With regard to fractures of the head of the radius, he was in complete agreement with Mr. Ogilvie. He did not regard excision of the head of the radius as a good operation with a movable elbow, as the elbow was very much weakened thereby; with a fixed elbow the operation was often advantageous.

He did not regard the important complete fracture of the capitellum as a rare injury, and operation in those cases was the only treatment. He also thought that the fractured internal epicondyle was not operated upon nearly enough.

With regard to the ideal position of ankylosis of the radio-ulnar joint, three-fourths pronation had been his practice for a long time in adults, and the correctness of that was borne out by Mr. Ogilvie's anatomy.

Mr. Watson Jones said he had looked up his records of minor elbow injuries treated during a two-year period in his fracture clinics at the Liverpool Royal Infirmary. The series included the following cases:—

	Number of cases	Age groups (%)				
		1-10	11-20	21-30	31-40	Over 40
Traumatic synovitis	94	9	*63*	14	9	*5*
Fracture head of radius ...	80	2	*30*	*21*	*25*	*22*
Fracture internal epicondyle	71	16	*78*	6	—	—
Fracture coronoid	28	3	*60*	14	13	10
Supracondylar fracture ...	63	*49*	*42*	2	3	4
Dislocation elbow	92	7	*45*	18	2	28

The Stiff Elbow.

The age-grouping merely proved statistically that which we all knew by experience. But it also emphasized the fact that it is the second decade in which elbow injury was most common and most significant. The elbow-joint of a young adult is very curious—it was peculiarly susceptible to injury, and stiffened on the slightest provocation even in the absence of bone injury. Fortunately this stiffness was not permanent. Examination after twelve months in the routine follow-up clinics showed that in four of the ninety-four cases of traumatic synovitis there was limitation of extension by 10° or less, and that in the other ninety cases the joint was indistinguishable from normal. The problem, therefore, was not how to cure these cases—they are all cured—but how to cure them quickly. Which treatment was most likely to reduce the incapacity period? In one clinic massage was used, in another massage and movements—active movements, assisted active movements, passive movements, manipulation under anæsthesia—all of these methods might be seen in various clinics; and a surgeon often continued to use one or other method, without knowing whether the treatment was doing the patient good or harm, because he had not measured and recorded the range of movement week by week. He (the speaker) would lay it down as a first principle that whatever method was used,

accurate measurements should be recorded. The method of treatment did not matter, so long as it was changed as soon as it was found that the range of movement was stationary or decreasing. He believed that if these records were more constantly kept, it would be more generally agreed that in the treatment of the stiff elbow, massage did no good, assisted active movements were liable to arrest progress, passive movements were definitely harmful, and manipulation under anæsthesia disastrous. In his opinion the quickest way to restore movement to the elbow stiff after minor injury was to do nothing.

Myositis Ossificans.

There were two generally accepted beliefs concerning myositis ossificans—(*a*) that it was caused by early passive movements; (*b*) that all movement must be arrested until the skiagram showed consolidation. Since this might involve four or five months of immobilization in flexion, conventional treatment had almost always caused permanent limitation of extension by 70° or 80°. Was this prolonged fixation really necessary? It was true that passive movement was to be condemned at any time, but this did not imply that all movement was equally to be condemned. In his opinion, by steering a course midway between excessive movement and excessive rest, our results could be improved. After immobilization for a week or two longer than usual, these cases should be left to themselves, progress being rigidly controlled by weekly measurements. If instead of increasing by about 5° each week, movement was being lost, the patient was doing too much; but as a matter of fact the patient himself was a wonderfully sound judge as to the degree of movement which was safe. Fig. 1 showed a supracondylar fracture with slight displacement, six weeks old. It had been treated throughout by passive movement, and myositis ossificans had developed. The passive movement was stopped at once, but the patient's own movement was not stopped. Measurements showed a steady increase of range, and the end-result (fig. 2) demonstrated not only as perfect a consolidation as could be secured by any conventional treatment, but also a normal range of extension. By clinical examination the arm was indistinguishable from normal.

Fracture of the Head of the Radius.

There could be no doubt that the prognosis in those slight, apparently insignificant marginal fractures of the head of the radius was much worse than the prognosis in supracondylar fractures of the humerus with the most gross displacement.

RESULTS OF TREATMENT.

1.—*Fractures of the Head of the Radius.*

	Number of cases	Results perfect	Loss of movement (average, in cases with imperfect result)
Without displacement	42 ...	93% ...	10°
Marginal and comminuted (treated conservatively)	22 ...	54% ...	25°
Marginal and comminuted (treated by excision) ...	10 ...	none ...	15°
Neck of radius with angulation (treated by manipulation or open reduction)	5 ...	60% ...	10°

2.—*Supracondylar Fractures.*

Without displacement	19 ...	100% ..	—
With displacement	41 ...	85% ...	25°
Comminuted (with involvement of articular surface)	3 ...	none ...	35°

Perfect results could be secured in 100% of supracondylar fractures without displacement, and in 85% of all supracondylar fractures, but in fractures of the head of the radius without displacement only 93% of the results were perfect, and in fractures with displacement less than 50%. Although in these cases, operation once

PLATE I.

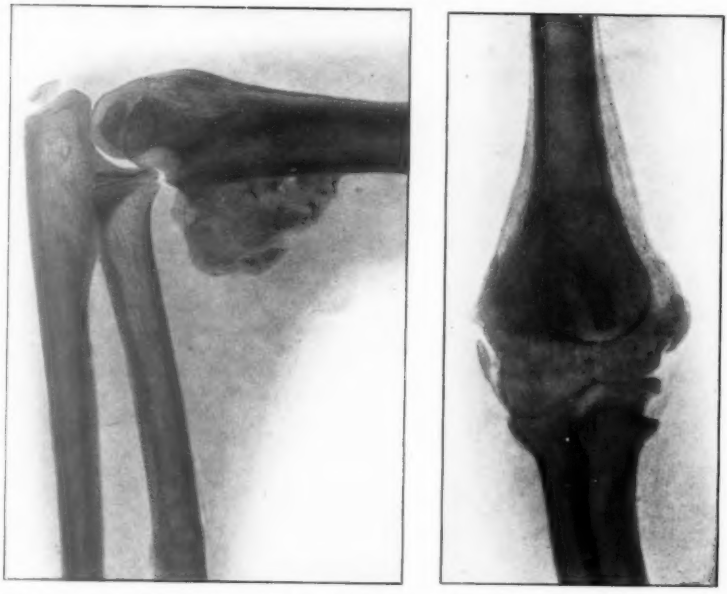

FIG. 1.—Supracondylar fracture of six weeks' duration. Myositis ossificans due to treatment by passive movements.

FIG. 2.—End result in same case as fig. 1. Although passive movements were stopped at once, no immobilization was used. Excellent consolidation of the new bone was associated with perfect range of movement. Clinically indistinguishable from normal.

decided upon, was carried out within a few days of the injury, the results represent fairly conservative treatment. It was not his practice to excise the whole head of the radius unless it was extensively comminuted or grossly displaced, because the shortening of the bone which inevitably followed this operation not infrequently caused secondary subluxation or dislocation of the lower end of the ulna, which was now relatively long. But more than this, he only excised marginal fragments when they had been displaced into the joint. Possibly the results would have been improved if marginal fragments had been more frequently excised, because although there was no perfect result after operative treatment, the final limitation of movement was much less in those cases treated by operation, than in those treated conservatively. It is interesting to note in the final results, that however treated, marginal fractures only caused limitation of extension, and did not interfere with radio-ulnar movement, because the sector of the head of the radius which was most exposed to trauma did not articulate with the ulna.

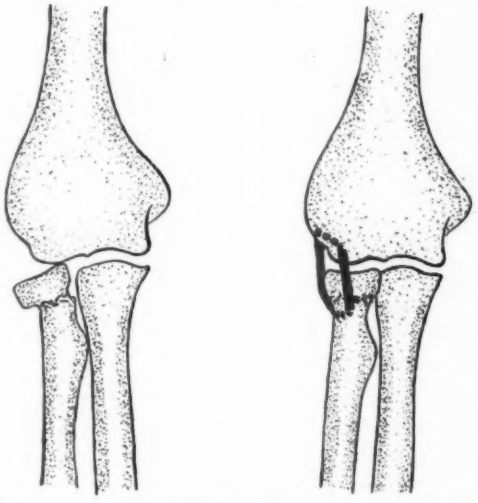

FIG. 3.—A "sling" operation for greenstick fracture of the neck of the radius with forward and outward tilting of the head.

On the other hand greenstick fractures of the neck of the radius with angulation of the head, always interfered with pronation and supination since the whole axis of articulation was altered. For this reason he had always regarded correction of such angulation as important. Since this injury occurred in children it was particularly important to avoid excision of the head, and if manipulative treatment failed, it was his custom to cut down on the bone from the outer side, to push the head into position with the assistance of a hook round the neck below the level of fracture, and to maintain correction by a sling of kangaroo tendon or fascia which passed in front and to the outer side of the head (fig. 3).

Injury to the Internal Epicondyle.

Ætiologically these injuries could be divided into those produced by direct trauma, and those produced indirectly by traction through the common flexor origin in falls on the outstretched hand. This distinction had very definite clinical

significance. It had been suggested by Platt,[1] that when internal epicondylar injuries were complicated by ulnar palsy, the nerve lesion was produced by contusion by the same blow that fractured the bone. But in an analysis of 71 cases, of which 17 were associated with dislocation other than outward, 9 with outward dislocation or subluxation, and 45 were isolated injuries, it was clear that ulnar palsy was only common in epicondylar separations produced indirectly by falls on the outstretched hand.

SEPARATION OF INTERNAL EPICONDYLAR EPIPHYSIS (71 CASES).

		Ulnar palsy
Associated with Elbow Dislocation. (100% Indirect Violence.)		
Outward dislocation	9 cases	7
Dislocation other than outward	17 cases	0
Unassociated with Elbow Dislocation.		
History of direct injury (80%)	36 cases	0
History of indirect injury (20%)	9 cases	1

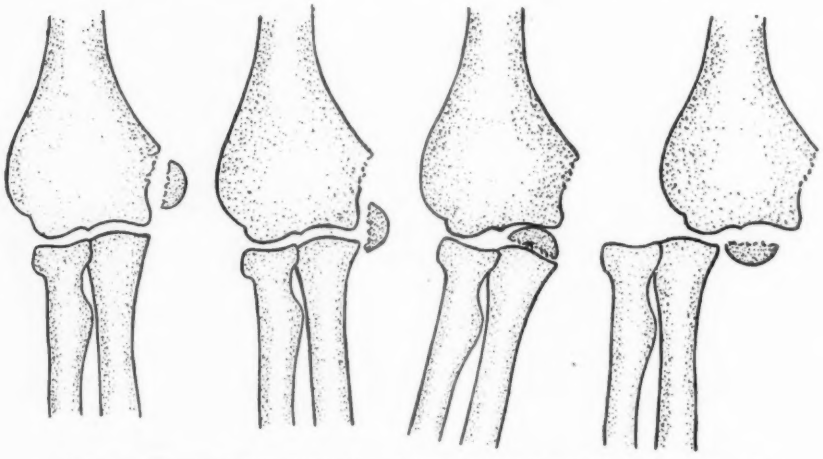

FIG. 4.—Diagram representing the four degrees of separation of the internal epicondyle produced by indirect violence. The degree of separation is an index to the degree of stretching of *all* structures on the inner side of the joint, and therefore an index to the frequency of ulnar palsy.

In other words, if a valgus strain on the elbow-joint was sufficient to tear off the internal epicondyle, it might also be sufficient to stretch the ulnar nerve and produce palsy. When there was no dislocation and the injury was due to direct trauma, palsy was rare. The solitary example of nerve complication in an epicondylar separation unassociated with dislocation, was in fact one of the 20% of cases in which the injury was indirect, and although the valgus strain had not been sufficient to dislocate the joint it had been sufficient to pull off the epicondyle.

Epicondylar injuries were, then, of two ætiological and clinical types (fig. 4)—the type due to direct trauma where the bone might be comminuted and in which ulnar palsy was rare, and the type due to indirect trauma—a fall on the outstretched hand. This second type was of four degrees, and the degree of displacement of the epicondyle was an index to the degree of stretching of all structures on the inner

1 Platt, H., *Brit. Journ. Surg.*, 1926, xiii, 51; "Robert Jones Birthday Volume," Oxford. Med. Publications, 1928, 195.

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PLATE II.

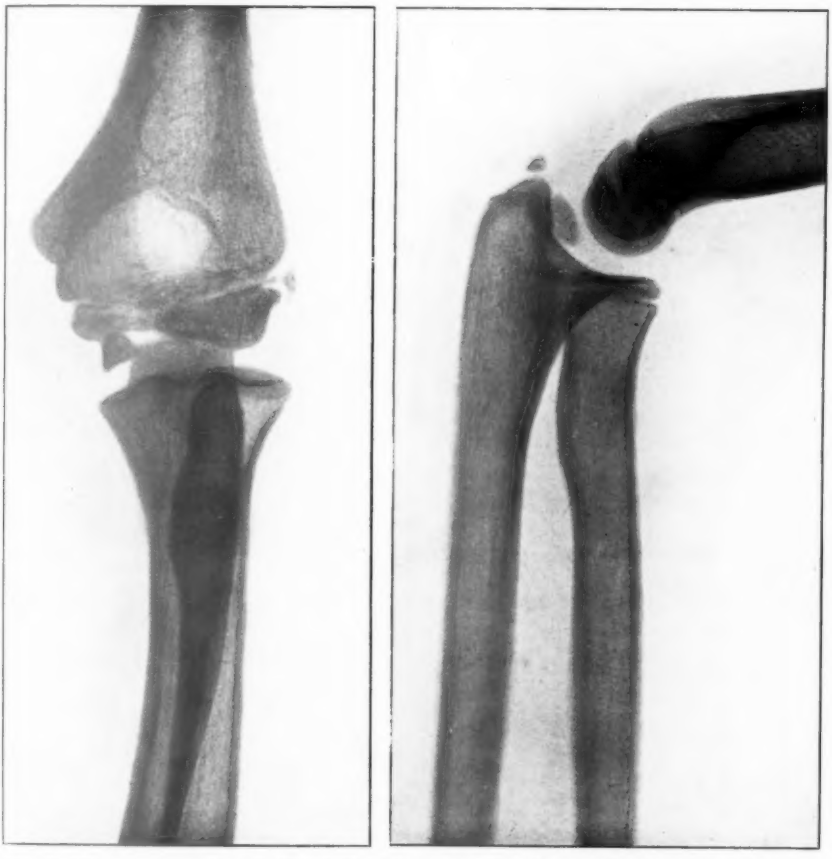

FIG. 5.—Displacement of the internal epicondylar epiphysis into the elbow-joint without joint dislocation.

side of the joint, and therefore, an index to the frequency of ulnar palsy. A slight strain produced slight separation and no nerve injury. A more severe strain produced considerable displacement and occasionally traction nerve injury. In the third degree the joint was actually prized open so that the fragment slipped in and maintained subluxation, and nerve palsy was almost always present. Finally, the force might continue sufficiently to dislocate the joint completely, and inevitably to produce a nerve injury unless there was also backward displacement so that the nerve could escape backwards from its groove. Any of these types might occur primarily, or on the other hand manipulation might convert the fourth into the third degree.

When the epicondyle was displaced into the joint (fig. 5), operative treatment was necessary for two reasons. Unless the fragment was removed from the joint, stiffness and osteo-arthritic change were inevitable. But more than this, the displacement of the capsule with the fragment produced traction on the mesentery of the ulnar nerve, and the twisting or kinking of the nerve trunk perpetuated the ulnar neuritis produced primarily by stretching. Cotton[1] had described a case in which the nerve itself was displaced into the joint. It was essential, therefore, not only to replace the epicondyle but to untwist the nerve, release it from its anchorage and either replace it in the postcondylar groove or preferably transpose it to the front of the joint.

Mr. B. Whitchurch Howell said his own experience of tennis elbow was that it was the result of bad tennis, that was to say, of not holding the wrist in sufficient dorsiflexion when performing the back-hand stroke. It was seldom that tennis elbow occurred in a professional player. Most of his patients had been of the arthritic type, and therefore some latent sepsis lurked in the tissues, to become manifest should any trauma occur.

He had under his care a patient who had a definitely cystic swelling in the region of Osgood's bursa, and who obtained complete relief by wearing a band below the level of the cyst. The swelling was oval in shape, and the size of one's little finger nail. He had another case, in which an operation had been performed elsewhere to find the cyst. It was not found. The patient had a strain along the musculo-spiral nerve going down to the region of the thumb. Because of the pain she had to give up hunting, as she could not rein back. The operation performed was removal of Osgood's bursa, but it had not cured her. The skiagrams showed a definite thickening on the external epicondylic ridge. That would prove that the pain experienced on pouring out tea, and also the pain in some cases of tennis elbow, was due to the traction of the extensors pinching the cutaneous nerve in that region.

He would show radiograms of two cases; the first was that of a man who was thrown off his horse and was dragged, not by the stirrups but by the reins. He probably ruptured the muscles in the region of the brachialis anticus, perhaps also stripping off the periosteum. There was marked traumatic myositis ossificans in the upper part, while in the lower part could be seen a spur. He supposed that the patient kept his elbow-joint flexed and taut in gripping the reins, and sustained muscular violence by that unusual method. He, the speaker, believed it was the only case of this type on record. There was limitation of flexion, but later there was practically complete flexion. The films of the pieces removed were also shown.

The next was a photograph of an elbow-joint in a tramway man. That man never had an injury of note, but on going up and downstairs on the tram he was often jerked, and held on to the tram rails to steady himself. In this way the muscles on the front of the elbow were kept constantly taut, and one day the joint was locked, and two loose bodies could be seen in the front of it. Both the injuries were minor ones, and were discovered through X-ray examination.

1 Cotton, *Journ. of Bone and Joint Surg.*, 1929, 348.

Mr. R. C. Elmslie said he would refer to two cases of "tennis elbow." The first was that of himself, but it was not the result of playing tennis, nor was the onset acute. Very few of the cases, he thought, came on acutely; often their commencement was so gradual that it was difficult for the patient to say exactly when the trouble began. He strongly associated himself with what Mr. Ogilvie said as to the toxic factor in tennis elbow. One seldom saw true tennis elbow in a young person, the subject was usually from 35 to 40 years of age, or older. It attacked the person who was playing active hard tennis after he had arrived at the age when most people had finally given it up. Many "tennis elbows" did not come on as a result of tennis at all. He had a man aged 72, who had not played tennis for many years. The speaker never played tennis in the winter, and his trouble commenced in the winter. He believed the cause in his own case was the wrenching of club-feet, admittedly a very strenuous occupation, comparable in intensity with tennis. Speaking from his personal experience he differed somewhat from Mr. Ogilvie as to the description of the symptoms. There were some very characteristic symptoms or signs in true tennis elbow; they were, first, the exact position of the tender spot, which was absolutely over the radio-humeral joint, not over the epicondyle nor over the head of the radius; rather it was between the two. Secondly, there were certain passive movements which cause pain, and they were characteristically forced flexion of the wrist and forced pronation of the forearm; and when it was more severe, forced extension at the elbow. The worst pain was that caused by a combination of the three: extension of the elbow, pronation of the forearm, and flexion of the wrist. A characteristic point was that it was brought on by active movements of extension of the wrist and supination of the forearm. The true tennis elbow was brought out in tennis by the fore-hand drive, getting the hand right back, extending the wrist and supinating the forearm. That was the position in which the pain was felt most acutely. He always excluded from the title "tennis elbow" cases in which there was definite evidence of arthritis of the elbow-joint. Here was a definite pathological cause for the condition, a cause which was absent in the ordinary characteristic tennis elbow. He was not saying there were not the same symptoms, but there were additional physical and radiological signs, signs that were not present in the usual tennis elbow. His own case of tennis elbow was cured by the removal of a molar tooth containing an apical abscess.

The second case he would not characterize as "tennis elbow," though it had similar symptoms. It was in a professional cricketer, and his symptom was inability to bowl throughout the summer season, because when he delivered the ball with his elbow extended it hurt him. It hurt him a little after an over or two, but after half an hour of it he was unable to bowl any longer. The X-rays showed slight signs of arthritis in the elbow, with some thickening on the posterior surface of the lower margin of the humerus. He explored the elbow at the back of the radio-humeral joint and found definite fringes. These fringes he removed, and also he chiselled off a little ridge at the back of the lower end of the humerus. The operation was done in July, 1928, and the same winter he bowled successfully in Australia and also through the present summer. That confirmed the view that there might be cases in which movements of extension and supination were painful in which fringes might be nipped in the extended position and so cause internal derangement of the elbow-joint.

With regard to physical treatment in injuries of the elbow-joint, it was the speaker's custom to teach that injuries of the elbow-joint, particularly in children and young people, gave the best and quickest results following normal and natural movements without assistance; that a child, whose elbow had been injured, would use that elbow and do the movements which were safe; but directly one started massage, assisted movements and passive movements, one risked doing something which was unsafe. Unfortunately, the present generation expected that injury would be treated by early massage and movements.

Mr. W. H. Trethowan contended that there was nothing more constant in its clinical picture than tennis elbow. The tenderness, he agreed with Mr. Elmslie, was in front of the joint. The joint at times was swollen both in front and behind, there was pain spreading over the extensor brevis, which was felt mostly on extension of the wrist in full supination. This summer he had himself been a sufferer; within two days he developed a bad tennis elbow, quite classical in its symptoms.

As the clinical picture was so constant, why was it thought necessary to imagine various pathologies? It was well known that bone-setters cured some tennis elbows by manipulation and failed to cure others. He also had had some miraculous cures from manipulation, but in some patients this treatment was unsuccessful.

In eight intractable cases he had explored the joint. He had found Osgood's so-called bursa in some cases; this bursa was nothing more than an extension of the synovial membrane of the radio-humeral joint, rather to the front of the external condyle. There was a variation in the amount of synovial membrane which projected forward, and the cause of the pain was the compression of this synovial pouch by the action of the extensor longior during the gripping action of the wrist. In the cases he had explored he had not found anything abnormal other than that all of them had, both in front and at the back of the joint, injected synovial fringes. He maintained, therefore, that tennis elbow was always a traumatic synovitis of the radio-humeral joint. He did not think that sepsis was a factor, as in all the eight cases simple removal of the fringes had sufficed to cure the condition.

The apparent inconsistencies of treatment depended on the stage at which the treatment was undertaken; as in the case of traumatic synovitis in any joint, whether rest, or manipulation or operation was required depended on the stage at which the case was seen.

Mr. Rocyn Jones said that in tennis elbow the lesion varied. Sometimes it would appear that the injury had occurred in the muscles, at others in the capsule of the radio-humeral joint; the uncertainty about its true nature was due to the lack of opportunity to investigate its pathology. There was reason to believe that the disability was produced by strenuous combined movements of the forearm, particularly that of flexion with supination, such as occurred when a low ball was attacked. The clinical signs were not always the same. In one group of cases there was distinct limitation of movement in certain directions, whereas in another there was no such impairment, but only pain when certain movements were carried out. In the former it frequently happened that when the forearm was pronated with the hand and fingers flexed, full extension at the elbow was limited, and any attempt to force extension was painful. In this type of case there were either adhesions present or some derangement of the radio-humeral joint, and manipulation under an anæsthetic to secure the full range of movement as advocated by Mills, was of distinct value. In the other group of cases where movement was fairly free, pain like that of neuritis persisted, or perhaps pain only in certain movements; in these the prospect of early cure was not nearly so good. Rest, radiant heat and massage often restored perfectly comfortable use to the forearm, but there occurred a proportion of cases that remained intractable, and for these an open operation offered the best chance of relief.

There was another injury of this region which might be confounded with tennis elbow in its clinical manifestation, and that was a fissured fracture of the head of the radius without displacement of fragments. The mechanism of production had been described by Mr. Ogilvie, and Mr. Rocyn Jones' experience of this injury confirmed this, for in almost all his cases the lesion was produced by a fall on the outstretched hand, the force of impact being transmitted along the shaft of the radius. Clinically, there was usually a tender elastic swelling over the radio-humeral joint and discomfort during pronation and supination movements; a radiograph revealed the

correct diagnosis. The only treatment necessary was resting of the forearm in a sling for a few weeks; massage and movements were inadvisable. When the fracture was healed, voluntary movements quickly restored full use to the limb.

Dr. Philip Figdor said he saw many cases of elbow injury, and active movements gave nearly 100% of good results. His chief trouble was to keep such patients away from people who would recommend forcible movements. Many of these cases were sent with direct instructions to give massage simply because the skiagrams had shown there was no fracture about the joint. He had seen some, after a few weeks' massage, develop myositis ossificans. Even in cases which had been mismanaged in the early stages, the patients, if allowed to carry out active movements of the elbow-joint, though there might be only a few degrees of movement possible, seemed, after many months to recover complete movement.

Mr. McCrae Aitken (President) said the debate had run almost entirely on "tennis elbow." Had time permitted, he would have ventured to disagree from nearly all who had spoken. Pain of a similar sort might be felt on the inner side of the joint, and this, he thought, was associated with the origin of the muscles arising from the internal condyle.

There were the kinds which were directly relieved by a pad and a strap, and there were others in which no pad or strap which he had devised was able to afford relief, but which on manipulation, "went click" and the patient was comfortable.

He cordially agreed with Mr. Watson Jones about the treatment of injuries of joints in the matter of massage and assisted and forced movements. In practically all cases the natural movements could be allowed to return with safety if the patient was safeguarded against indiscretions.

Section of Urology.

[*November* 28, 1929.]

Specimen of Testicular Carcinoma Metastasized from the Small Intestines.—ZACHARY COPE, M.S.

The patient was admitted to the Bolingbroke Hospital in 1920 with symptoms of acute intestinal obstruction. The abdomen was distended and there was a good deal of free fluid in it. I operated and found in the small intestine a ring stricture, such as is often found in cancer of the large intestine, and all I was able to do was to carry out a short circuit. The pelvis was filled with secondary growths, therefore excision was out of the question. The patient was so ill that it did not seem likely that he could live longer than three or four months. However, he survived the primary operation and his condition improved, but he afterwards went downhill for three months. Selenium was then tried, and nineteen months after the first operation I showed the case to the Medical Society of London as one which might have been benefited by selenium, since the patient had increased in weight to the extent of two stone. I said then that it was not proved that the selenium had had much effect, but it might have had. The man continued at work and I lost sight of him, and concluded that he must have died. However, eight years later I was asked to see him because of the condition of the right testicle. There was a nodule in the globus minor, which I diagnosed as tuberculous, and I thought a mistake might have been made in the primary diagnosis. I removed the testicle, and at the operation I put my finger into the abdomen once more, and found growth all over the pelvis and little nodules on the peritoneum further up the abdomen. I excised one, which is now shown. The microscope shows that the testicular and peritoneal nodules are secondary deposits of cancer of the small intestine. I believe this case to be unique. The carcinoma is of the type called "argentophile," and is similar to that which attacks the appendix. The growth must be of a mild type, as the patient has had secondary growths all over his abdomen for nine years, and he is now better than he was a few years back. How the secondary growth got there, whether by way of the blood-stream or the lymphatics of the spermatic cord, I do not know.

Dr. CUTHBERT DUKES said he had been much interested in these sections, as this was a kind of tumour which but rarely formed metastases. It occurred usually as a primary growth of the appendix or small intestine. Three years ago Stewart and Taylor, in a review of these tumours, recorded seventeen cases with metastases.[1] The same year Dukes and Lockhart-Mummery reported another case of a primary tumour of the ileo-cæcal region, with metastases on the peritoneum, liver and regional lymph-glands.[2] That was No. 18, and if no other cases had been reported since, the present case might be regarded as No. 19.

The origin of these tumours was a matter of considerable controversy. Masson thought that they arose from nerve cells. They grew very slowly. He had not heard of one in the testicle before.

Papilloma of Renal Pelvis.—ZACHARY COPE, M.S.—This patient had hæmaturia intermittently for eighteen months. Two cystoscopic examinations were made, one by myself, but at neither examination could it be ascertained from which side the bleeding came; there was no bleeding when the examination was made. Nephrectomy was performed when the side of the bleeding was determined. In one place the growth looked carcinomatous, but the pathologist said there was no trace of malignancy.

1 Stewart and Taylor, *Journ. Path. and Bact.*, 1926, xxix, 137.
2 Dukes and Mummery, *Journ. Path. and Bact.*, 1926, xxix, 308.

Nævus of Bladder.—SYDNEY MACDONALD, F.R.C.S.

This was excised in June, 1929, from the bladder of a male patient, about 33. The only symptom was a slight terminal hæmaturia, beginning about a month previously. Cystoscopy showed the tumour, apparently a nævus, growing from the left upper quadrant. Microscopically, the tumour is a cavernous angeioma.

Carcinoma of the Bladder arising in the Urachus.—A. CLIFFORD MORSON, F.R.C.S.

This specimen is of remarkable pathological interest. The patient is a man aged 44, with the usual symptom of a growth of the bladder, namely, a painless hæmaturia. The tumour was removed by the endothermy knife.

The section shows an atypical neoplasm containing colloid material. Ewing, the pathologist, states that colloid carcinoma never arises primarily in the bladder, but is always secondary to a growth in the intestinal tract. In this patient there seemed to be no evidence of a primary tumour in the bowel. Part of the urachus was patent and full of growth. The remainder of the urachus was about the size of the vas deferens, and extended from the umbilicus to the commencement of the growth. As the tumour was removed only a week ago, there has not been time to go carefully into the pathological side of the case, but it was believed to be a very rare specimen.

Postscript.—The patient, eight days after operation, suddenly became unconscious, and died on the eleventh day. At the post-mortem examination, performed by Dr. Richmond Lane, the primary growth, a chordoma, was discovered between the fifth lumbar vertebra and the sacrum. Death was caused by a metastasis in the occipital lobe of the brain.

Further details of the unique tumour will be published at a later date.

Pyelography in Renal Tuberculosis.—A. WILFRID ADAMS, M.S.

I am showing specimens and pyelograms of a series of four cases illustrating pyelography in different degrees of tuberculous invasion of the kidney.

(1) V. S., male, aged 23. Eight months scalding pyuria. Bladder urine contained pus and tubercle bacilli. Segregated urine showed pus only on both sides. Pyelography of both kidneys showed definite excavation of cortex of right kidney only, $\frac{1}{3}$ in. in diameter, thus supplying the requisite clue for the side requiring nephrectomy. Operation (April, 1929): The split kidney exhibited the cavity indicated by pyelography. Now (November, 1929), apart from the history, the patient's doctor would pass him as a "first-class life." No constitutional disturbance followed pyelography.

(2) E. F., female, aged 23. Four months' dysuria. Urinary segregation pointed clearly to tuberculosis of the right kidney, and pyelograms confirmed the presence of a large cavity in the upper pole of that organ. The right kidney was therefore removed. No constitutional effect from pyelography.

(3) M. S., female, aged 25. Six months' dysuria and lumbar ache. Tender lump in right loin and pyrexia. Pus and tubercle bacilli were present in both kidneys but a normal structure shown in the left pyelogram led one strongly to advise removal of the grossly excavated right kidney. This operation was carried out in October, 1928, and now (November, 1929) the patient is extremely pleased and is looking a picture of health. Increased pyrexia followed pyelography but did not prevent good primary healing of the wound.

(4) F. C., female, aged 38. Right-sided pain for many years. Severe cystitis and tubercle bacilli in urine (1914). Still pain and slight frequency—and partial incontinence—of urine at times (1928). Chromocystoscopy revealed left-sided efflux only. On the right side, the tip of the catheter lodged in a slight depression, presumably the vestige of an obliterated ureteric orifice. The left pyelogram was normal. At operation

a closed hydronephrosis was removed on the right side. The pathologist reported that the ureter was a fibrous cord with no lumen. Possibly it is a case of obsolete tubercle.

Pyelograms of Two Tuberculous Kidneys.—E. T. C. MILLIGAN, F.R.C.S.

It is not my practice, solely on theoretical grounds, to do pyelography of tuberculous kidneys, sufficient data for diagnosis and treatment being available without this extra manipulation.

These two cases necessitating pyelography were somewhat unique, in that they were both regarded, over many months, as examples of *Bacillus coli* infections of the urinary tract; in fact this organism was found and held responsible for the pus in the urine.

At my first clinical examination of both cases, tuberculosis of one kidney and bladder was suspected in each case, but, again, tubercle bacilli could not be found in the urine and *Bacillus coli* was present. Attention was directed to the tuberculous kidney in both cases by the indigo-carmine excretion test.

The reason for showing these pyelograms and for asking Mr. Adams to exhibit his series, is that they might be helpful to those who are afraid of investigating tuberculous kidneys in this way and to whom such pyelograms are unfamiliar.

It is of interest that in addition to the almost closed focus of tubercle in the lower pole, accounting for the absence of tubercle bacillus in the urine on so many occasions, and found only when the pelvis was dilated with opaque fluid which allowed the cavity to evacuate itself, there was dilatation of upper unaffected calices; in fact, hydronephrosis in each case.

In both cases there were symptoms of associated hydronephrosis, attacks of loin pain, and in one case pain and temperature after exertion.

One would expect the hydronephrosis to be due to the thickened infected ureter but, strangely enough, the ureter was not obviously thickened, as is usual in these cases, indeed the pathologist had some difficulty in finding infiltration of cells.

In both these cases, not an uncommon occurrence in tuberculous ureters, the ureteric catheter was arrested a few centimetres up the ureter on two occasions. On the third attempt a very small catheter was passed, which enabled pyelography to be carried out.

Case of Stenosis of the Left Ureteric Orifice following (in one year) Removal of an extensive Villous Papilloma of the Bladder by Open Diathermy. Pyonephrosis. Nephrectomy.—CLAUDE H. MILLS, M.R.C.S.

July 6, 1928.—E. M., male, aged 61, had been attending St. Paul's Hospital for the previous six months, undergoing a course of arsenobenzol and bismuth treatment for an old-standing syphilitic infection of thirty-eight years' duration, presenting no clinical signs other than a persistent Wassermann reaction (++). Patient was transferred to me on account of left-sided renal pain with frequency of micturition and pyuria. He gave a history of having had a tumour removed from his bladder almost exactly a year ago at King's College Hospital. From the Registrar of that hospital was received the following report:—

"E. M. was admitted to this Hospital (K.C.H.) on July 6, 1927, under Mr. Everidge. Cystoscopy was performed on July 8, 1927, when an extensive villous tumour was seen encircling the neck of the bladder, too extensive for fulguration. At cystotomy on July 11, 1927, a large papilloma was removed by means of the diathermy knife. The patient made a good recovery. Histologically the tumour was a simple papilloma with no evidence of malignancy."

Ever since the above operation the patient has had nocturnal frequency of micturition, and during the past six months there has been considerable pyuria with periodic attacks of left-sided renal pain. Six weeks ago he was in bed for twelve days with a severe attack.

Reflexes normal. Cerebrospinal fluid non-pathological.

July 27, 1928.—On palpation, neither kidney appeared to be enlarged, but deep pressure was definitely more painful on the left side.

Cystoscopy showed moderate trigonitis; no trabeculation, sacculation, stone, or growth. On the left side of the trigone, extending upwards and outwards from the internal meatus, was a pale, puckered, cicatrix resulting from the removal of the growth by diathermy one year previously. The left ureteric orifice could not be localized, the right looked normal with a clear efflux, and intravenous indigo-carmine appeared from it in 4½ minutes. No indigo-carmine was seen from the left in 25 minutes.

August 3, 1928.—During vigorous massage to the left renal area a very fine coil of pus was seen to exude from the left ureteric orifice, which was almost "pin-hole" in size. Attempts to introduce even the finest ureteral catheter were unsuccessful.

August 13, 1928.—Attempts to enlarge the left ureteric orifice by diathermy were unsuccessful owing to the inability to locate it again with intravenous indigo-carmine, a ureteral catheter being meanwhile retained in the right ureter to keep the visual field clear. No dye appeared from the left ureteric orifice during 45 minutes' observation.

September 28, 1928.—A pyelogram of the right kidney indicated early hydronephrosis, as there was slight clubbing of the minor calices and the pelvi-ureteric angle was 90°. However, the urine was sterile, and the function tests satisfactory. Repeated radiograms failed to show a shadow of the left kidney.

Operation, January 18, 1929.—Through a lumbar incision I removed the left kidney, together with a considerable portion of dilated ureter. The sacculated kidney was very adherent to its fibro-fatty surroundings. The patient made a good recovery.

Routine Cystoscopy, November 8, 1929, *i.e., ten months later.* — A small papillomatous recurrence was destroyed by diathermy in the scarred area on the left side of the trigone. There was no cystitis.

My object in describing this case is to emphasize the importance of frequent routine cystoscopic examination of cases in which excision of the bladder wall in the neighbourhood of the ureteric orifices has been performed, not only for the early detection of possible tumour recurrence, but also to deal with stenosis of the ureteric orifice from scar contraction, should such eventuate.

When one considers the increasing number of treated cases in which the removal of a vesical growth has necessitated excision or destruction of mucous membrane in the proximity of the ureteric orifices (more especially as since the introduction of surgical diathermy, the ureter is less frequently reimplanted), the necessity of impressing upon all such patients the importance of subsequent observation is obvious.

Specimen of Large Hydronephrosis.—E. T. C. MILLIGAN, F.R.C.S.

This large right hydronephrotic sac, 26 by 26 cm. by 12 cm., is shown because sacs of such magnitude are not often seen nowadays.

The patient, aged 70, and her medical advisers had been content for many years with the diagnosis of ovarian cyst, and she had tolerated the greatly increased size of her abdomen until she began to pass blood in the urine, when an independent lesion of the kidney was suspected.

On two occasions cystoscopic examination revealed blood coming from the right ureter from which no indigo-carmine was excreted. A ureteric catheter was twice arrested at 6 cm.

The patient, wearied of these examinations, ceased to attend until bleeding was again alarming. On the next occasion a very small uretereric catheter fortunately passed to full length, and X-rays showed it curled up inside the tumour, proving

that the growth was not an ovarian cyst. A pint and a half of blood-stained urine was drawn off through the ureteric catheter. It is difficult to say why it was blood-stained, for no growth or inflammation was found in the tumour.

The patient was averse to operation, but later, as blood-stained urine continued, she sought relief.

The removal of such a large cyst was made possible and easy by the facts that she was a thin woman and there were no firm adhesions to surrounding structures.

The tumour extended to the opposite side of the abdomen, down to the pelvis and up to the diaphragm. It proved an easy task to separate, deliver and remove this immense swelling.

Two Cases of Pyelovenous Backflow.—CYRIL NITCH, M.S.

I.—Female, aged 51. Relapsing *B. coli* cystitis for about twelve years. Never had symptoms of pyelitis.

Cystoscopy in October, 1929, one month after beginning of another attack of cystitis. Moderate basal inflammation; ureteric orifices appeared normal and were catheterized without difficulty. Pure culture of *B. coli* from bladder urine; urine from kidneys normal and sterile. Pyelograms made with 8 c.c. of 13% sodium iodide. Injections made very slowly with a syringe and stopped immediately patient complained of discomfort in the loin. Right kidney injected and X-rayed first. Ureter kinked just below renal pelvis; calices slightly clubbed; clear shadow of pyelovenous backflow throughout kidney. Left kidney injected and radiographed five minutes after right. Pelvis and calices normal. Both kidneys included in film, but all signs of backflow in right kidney had disappeared. Patient suffered no inconvenience or ill-effects.

II.—J. M., male, aged 51. Impacted oxalate calculus removed from lower end of left ureter thirteen years ago; three attacks of right renal colic during last ten years. The last, a month ago, was very severe and lasted two days. Probably a small stone was passed, as none was visible in skiagrams.

Cystoscopy (*October*, 1929).—Bladder and ureteric orifices normal. Bladder urine contained few pus cells; left kidney urine normal; right contained casts.

Pyelography: 10 c.c. of sodium iodide injected slowly with syringe. Injection stopped immediately patient complained of discomfort. Left pelvis deformed and ureter dilated. Right pelvis slightly dilated, calices clubbed, ureter dilated and kinked an inch below pelvis. Well-marked pyelovenous backflow in right only. No ill-effects.

Pyelograms of a Double Ureter and Double Pelvis.—CLIFFORD MORSON, F.R.C.S.

This is an interesting example of Nature's extravagance in trying to produce a third kidney. There are two ureters on the right side, each of which I catheterized and injected with a solution of sodium iodide, thus showing two pelves. It will be seen from the illustration that the lower pelvis has calices, whereas the upper pelvis has none (fig. 1). Two or three years ago I showed before the Section a specimen of a kidney which it had been necessary to remove because of retention of urine, infection, etc., in the case of an infant aged nine months. The pelvis was perched on the top of the upper pole, and in the discussion on this point I said that no complete third kidney had yet been found in the human body. That statement, however, was not quite correct. In no living human subject had a third kidney been found which was completely developed, but the Professor of Anatomy at Trinity College, Dublin, informed me that he had removed a third whole kidney post-mortem, and sent me the literature. Here are three ureteric buds, one on the left side and two on the right, and from these are developed the

renal pelves and calices. Yet the mesonephros, which produced the cortical part of the kidney, has failed, as it always does in these cases, to fall into line with the excessive development of the ureteric bud. There seems to be no coördination between the ureteric bud development and the development from the mesonephros.

FIG. 1.

FIG. 2.

FIG. 1.—Pyelogram of right kidney showing the lower of the two pelves with defective development of the calices. Note the shadow of catheter in the accessory ureter. (Mr. Clifford Morson's Case.)

FIG. 2.—Pyelogram of right kidney showing the upper of the two pelves with total absence of calices. Note there is no communication between the two pelves. (Mr. Clifford Morson's Case.)

Two Specimens of Double Ureter.—JOCELYN SWAN, M.S.

I.—The first patient was a woman aged 50, who came under observation about six months ago with a history of an attack of painless hæmaturia which had lasted for three days during the previous week. When she presented herself, the urine was free from blood and albumin, and on routine examination by a cystoscope, two ureteric orifices were seen on the left side and one on the right. After an intravenous injection of indigo-carmine, both orifices on the left side and the single orifice on the right showed a strong efflux of coloured urine after an interval of five minutes.

FIG. 1.—Pyelogram of double ureter and double renal pelves on the left side in Case I. (Mr. Jocelyn Swan's Case.)

FIG. 2.—Composite skiagrams of right and left sides in Case II, obtained by super-imposing skiagrams of each side. (Mr. Jocelyn Swan's Case.)

Ureteric catheters were passed into the two orifices of the left side and a pyelographic examination was carried out, at which it was found that there were two distinct pelves, but whereas the lower pelvis was of normal shape and had well-marked calices, the upper pelvis was slightly blurred and the upper calix turned inwards. I am inclined to think that some pathological lesion is present in the upper pelvis of the left side, but in the absence of any further bleeding and on such slender evidence, I did not feel justified in exploring the left kidney.

II.—The second case is that of a man aged 46, who also complained of an attack of painless hæmaturia lasting twenty-four hours, during the previous week. Nothing could be felt on clinical examination, and on cystoscopic examination after an injection of indigo-carmine, a perfect bombardment of coloured effluxes was seen in the bladder, coming from four distinct ureteric orifices within six minutes after the injection. There were two orifices on each side, these being situated close together on a marked inter-ureteric bar. An attempt was made to pass catheters into all four ureters, but although this was accomplished with three catheters, the fourth could not be introduced. The two sides were therefore examined on separate occasions.

On the left side the lower pelvis was normal, and showed well-marked minor calices, but the other pelvis was small, the calices were irregular, and the upper calix appeared to be on the inner side of the pelvis. On the right side a similar appearance was obtained, the upper pelvis again showing an internal calix.

It is usual that when two complete ureters are present on one side, the lower ureteric orifice in the bladder leads to the upper pelvis of the double kidney. This is true in the first case, and was also true of the left side in the second case, but on the right side of the latter the lower ureteric orifice leads to the lower pelvis of the double kidney. This patient has had no further attack of bleeding, and with the good colour efflux from each orifice and from the similar appearance in the pyelographic examination of the two sides, there is no evidence to show from which side the bleeding had occurred, but I hope to have the opportunity of again examining the patient during an attack of bleeding.

Mr. CYRIL NITCH said that in one case of double ureter, which he had seen in a female patient, there had been continuous hæmaturia for several weeks. The efflux on the right was deeply blood-stained. A ureteric catheter was passed 5 cm. and withdrew blood-stained urine; it was then passed as far as it would go, and the efflux became quite clear. On its withdrawal for some distance, blood-stained urine again escaped and continued to do so when the catheter was again passed as far as it would go. He thought the case was one of papilloma of the ureter, and that the blood-stained efflux on passing the catheter its whole length the second time, was due to trauma. The urine from the left kidney was normal. He exposed the right kidney and found that it had two apparently normal ureters, one rising from a normally placed pelvis and the other from a small pelvis in the lower pole. The two ureters were small, and they united to form a normal-sized ureter, about three inches below the kidneys. The kidney was incised and each pelvis carefully examined. The upper was normal, but the lower was blood-stained. There was no macroscopic evidence of angeioma. He removed the kidney, as hæmorrhage had been severe for three weeks, but, to his consternation, he found no naked-eye cause for the hæmorrhage. Professor Dudgeon reported chronic interstitial nephritis with blood-pigment, blood, and blood-casts in the renal tissue. Before the operation the urea concentration was normal and several examinations for casts proved negative. Four years later the patient was well and there had been no recurrence of hæmorrhage. This was one of the causes of hæmaturia in double ureters.

Section of Balneology and Climatology.

President—Dr. G. L. Kerr Pringle, M.C.

[*November* 15, 1929.]

PRESIDENT'S ADDRESS.

(Abridged.)

A Summary of Two Thousand Consecutive Cases of Rheumatic Disease.

By G. L. Kerr Pringle, M.C., M.D.

In taking notes, one is apt, as time goes on, to pay attention to certain points which in the earlier cases one had not particularly noted, and when a survey is made of a number of cases, points which one now considers important have been found to be omitted in the earlier cases. This is inevitable as one's views become enlarged and alter. There are many advantages in reviewing a large number of cases. Opinions which may previously have been held may, on examination, be found to be unreliable. For instance, psoriasis is said to be a common accompaniment of rheumatism, yet I only find mention of 17 instances. Again, points which one had not considered of importance are brought into evidence.

Table I shows the number of cases.

I have used the classification of the Ministry of Health with subgroups in the arthritic series (Table II).

There are no cases of acute rheumatic fever in the series. I have, however, added a sub-group "chronic rheumatism," cases which I believe cannot be called subacute and which are not fibrositis or arthritis. It is difficult to determine when subacute merges into chronic rheumatism, and I am not convinced that chronic rheumatism has always a subacute stage. There are patients with a history of rheumatic fever in childhood who, years after, develop a condition which is not fibrositis, a rheumatism of muscles and fascia associated with a dry condition of joints which is hardly arthritis. This may be the variety of arthritis which Strangeways called the "dry" type. In the meantime I use the term chronic rheumatism, as it is not confined to the joints.

Subacute Rheumatism (Table III).—Seventy-two cases had a previous history of an acute attack of fever with definite heart involvement in 39, and chorea in 4. Septic tonsils were present in 33 cases, 29·7%. Where there is evidence of septic tonsillitis, I have the tonsils enucleated at the first opportunity, because foci which may appear inoperative may be made operative by severe illness. Dental sepsis was present in 11 cases.

Chronic Rheumatism (Table IV).—There is a history of rheumatic fever in 29 cases, and of dental sepsis in 10 cases. Probably this figure is really much higher. We have a rule in the Royal Bath Hospital that the patients' mouths must be put in order before admission. I have considerable doubts about this rule, as one is entirely in the hands of some dental surgeon who may do a wholesale clearance at one sitting, which may be justifiable in a robust individual but which

may lead to disaster in a debilitated subject and produce an exacerbation of the disease. It is wiser to remove one or two teeth and then wait a week before removing more.

I believe that the intestine, particularly in conditions of stasis and ptosis, plays an important rôle in these cases. Such cases do well with aperient waters and when lavage of the colon is employed.

Fibrositis (Table V).—A history of rheumatic fever in 36, and enlarged thyroid in 13. Dental sepsis was present in over 25% of the cases, and artificial menopause in 15 of the women.

FIG. 1.

Occupation : In this survey only the occupation of the hospital patients is given. Among the males the largest proportion occurred amongst outdoor workers. Among the females, excluding house-workers, 21 were engaged in textile factories.

In close relation to the fibrositic group come the next two groups, neuritis and sciatica.

Neuritis (Table VI).

Sciatica (Table VII).—History of rheumatic fever, 4 ; dental sepsis, 25.

Occupation : Amongst the males the larger number were engaged in outdoor work.

Proliferative arthritis (Rheumatoid) (Table VIII).—This, I believe, is associated with a septic focus. Clinically, I recognize at least three types in this group: (1) The ordinary adhesive form, leading to rapid ankylosing of the joints; (2) the non-ankylosing type in which there is always free movement of the joints, and a history of distension of the capsule due to fluid, with subsequent absorption. Subluxation may occur, but there is no ankylosis; (3) variety in which there is little or no alteration in the bones, but marked fibrous contraction and the finger

FIG. 2.

joints become flexed and the hand becomes fist-like. This variety would appear to be similar to that described by Jaccoud [1].

I have at present under my care still another clinical variety, which I do not recollect having seen before. The X-ray appearance is very similar to that in the atrophic form (figs. 1 and 2). This patient is a man aged 34, and the condition started in March last. Finger-joints, wrists, elbows, knees and ankle-joints are all affected. There is distension of joint capsules, and I have twice aspirated the fluid, which

is turbid and contains pus, but we have not been able to culture it. The unusual point in this case is that in all the affected joints you can rub the bone surfaces against each other, producing a grating feeling, quite distinct from anything I have ever felt before.

Another clinical type is that form associated with the name of Poncet [2]. I have one case not in this series which I think is definitely tuberculous. The patient, a young woman aged 33, has, in addition to arthritis of the knee-joint, well marked

FIG. 3.

Bazin's disease of the skin of the calf. The joint is now quiescent, and her general condition has improved. Treatment has been mainly that of injections of arsenic.

Occupation : Indoor workers are most frequently attacked, especially among the females.

Atrophic arthritis (Table IX).—Douthwaite [3] gives a good definition of the condition as a "disease occurring largely in females of reproductive age, and characterized by little or no pyrexia, low blood-pressure and unstable pulse-rate, scattered pigmentation of the skin on a basic waxy pallor, generalized wasting in addition to pronounced atrophy of the muscles about the affected joints, viscero-

ptosis, hyperchlorhydria, exaggerated tendon reflexes and symmetrical swellings of smaller joints, particularly those of the fingers. The skin over the affected articulations is pale and atrophic, and local tenderness to pressure is never a striking feature." Strangeways [4] believed this variety to be limited to women. X-ray appearances show marked loss of lime salts in the neighbourhood of the articulations, and there is definite stencilling of the outlines of the bones (figs 3 and 4).

Six had rheumatic fever in childhood, 11 had abnormal menstruation or leucorrhœa.

FIG. 4.

In seven the condition immediately followed pregnancy; and previous enlargement of the thyroid was noted in seven cases. Achlorhydria was only noted in two cases, but many of these cases came under observation before Hurst drew attention to the presence of achlorhydria, and therefore the figures are not complete. I believe that these cases are metabolic in origin and probably due to some endocrine system imbalance.

Occupation: All the patients were indoor workers.

Osteo-arthritis (Table X).—A skiagram of an osteo-arthritic hip not in this series shows what appears to be an incomplete fracture of the neck (fig. 5). I had two cases of this description within a week. Both were men, aged between 50 and 55, and both had suffered various traumas on the hunting field. The X-ray appearance in the second case was exactly similar to that in the first. What is the prognosis in these cases?

Occupation: Outdoor workers were in the majority.

FIG. 5.

Chronic villous arthritis. Climacteric or menopausal arthritis. (Table XI.)—The majority were obese. Fibrositis, panniculitis and Heberden's nodes were present in a large number of cases. If not treated in the early stages, the joints tend to become osteo-arthritic.

Gout (Table XII).—*Incidence of Previous Attack of Rheumatic Fever* (Table XIII).—I wish, in this survey, particularly to bring to notice the incidence of a history under this heading. Excluding the 72 cases belonging to the subacute rheumatic group, there are 130 cases, or 6·8%, among the other groups. This is a fairly large percentage. I believe it must be considerably more, as it was only about

half-way through these series of cases that I became alive to the frequency with which it occurred. It is rather difficult to extract information as to whether the patient really had rheumatic fever or not. Many patients will tell you that they "just escaped rheumatic fever," whatever that may mean. I inquired into the length of the illness, the length of time they were kept in bed, the presence of fever, sweating, condition of heart, and history of chorea.

In looking through these cases, two or three points were brought to my notice. Psoriasis has already been referred to. The occurrence of asthma in certain types of arthritis was of some interest. There are only eight cases mentioned, six occurring in the proliferative group.

Again, I have for some time been interested to note that a surgical or artificial menopause was a fairly frequent accompaniment of arthritis in the female.

These tables show that:—

15	cases	occurred	among	361	women	in the	fibrositic group
10	,,	,,	,,	325	,,	,, ,,	proliferative or infective group
11	,,	,,	,,	160	,,	,, ,,	chronic villous group

Occupation: (Males) out of 566 cases, 335 were engaged in mining, labouring, outdoor employment and the engineering trade, in which occupations they are exposed to changes in temperature, exposure and trauma. 139 were definitely employed indoors, and in 92 the occupations were partly indoor and partly outdoor or they were past work and pensioners. The trade of butcher is only represented on four occasions; (Females) all the patients were indoor workers, with the exception of two Church workers.

Treatment: You cannot have efficient satisfactory treatment without accurate diagnosis and, to obtain this, time must be spent on the previous history and an attempt made to classify the type of case. There is no royal road in the treatment of diseases of this group. It is bad business to treat a case of menopausal arthritis by the same methods you would adopt in a case of proliferative arthritis, and vice versa. Treatment must not stop with the removal of an infective focus; the treatment, as far as we physicians are concerned, is then only just commencing. Our duty is to try to build up the patient's resistance and to restore the affected joint, if possible, to normal. Unfortunately we so seldom see the patient until considerable damage has been done, much time having been wasted by treating the patient with salicylates; and this method of treatment still goes on in spite of the abundant fresh literature on chronic rheumatic diseases.

Treatment may be divided into five heads: (1) Removal, if possible, of presumable infective foci; (2) medicinal; (3) physiotherapeutic; (4) dietetic; (5) orthopædic.

(1) Infective foci: Dental, nasal, tonsillar, prostatic, uterine and intestinal. The teeth and tonsils I have dealt with.

Cumberbatch and Robinson have opened up a new field of treatment by the application of diathermy directly to the prostate and uterus, and in the Harrogate Royal Bath Hospital we have already had some gratifying results.

Intestinal cases are much more difficult to assess. What is the normal flora of the intestines? Bacteriologists are unable to tell us, and to believe that the presence of a *Streptococcus viridans* in a bowel-washing indicates cause of an arthritis, requires considerable faith. Cases do undoubtedly improve with intestinal lavage, but by washing out the bowel you are relieving the stasis which is so often an accompaniment.

Medicinal.—Iodine, preferably the French tincture, in doses up to 15 mins. t.d.s. is the best; its action is, however, slow, and it must be given for long periods. It is specially useful in the fibrositis associated with proliferative arthritis, and in menopausal arthritis.

When there is debility, arsenic is a useful drug given hypodermically, as the cacodylate of soda; especially would I recommend it in the atrophic cases.

Thyroid gland extract, combined sometimes with pituitary and ovarian extract is useful in the atrophic and menopausal variety. If tachycardia is present I give $\frac{1}{60}$ gr. of digitalin along with the thyroid. For pain and restless nights, then 10 gr. of calcium acetosalicylate along with 10 gr. of a bromide salt are given each night.

Sulphur is distinctly useful. There are many instances of patients with chronic rheumatism who have received immense benefit from drinking the old sulphur-well water without taking any baths. This is not simply due to its aperient effect, as Dr. David Brown has shown us from his laborious investigations.

Physiotherapeutic.—The various methods of physiotherapeutics are well known to you. I have a great belief in the action of contrast baths. The rheumatic as a rule allows his skin to go out of action, he keeps it at one temperature; he has been told to keep warm and wear flannel, and not expose himself to draughts, and the consequence is that his skin never reacts, and his cutaneous circulation becomes stagnant. We give patients hot baths, either with dry or wet heat, to produce an arterial hyperæmia, and we often stop there; whereas it is much better to follow up this hyperæmia by employing what has been described as a "metabolic whip," a tepid or cold shower or douche. A much better reaction is thus obtained and the patient is acclimatized to rapid changes of temperature. And if, as we believe, the weight is in favour of the presence of imperfect oxidation in these cases, what better method have we of stimulating metabolism? Of course this must be done gradually, much depending on the physique of the patient.

Massage.—Much harm may be done by massage. Intelligently ordered and intelligently carried out, it is excellent and can be productive of much good. The fault lies mainly with the practitioner who simply orders massage without details, and the manipulator is left to his own devices. Some have judgment and some have not. Often dry massage is started too early, or it is given too frequently.

Diet.—Much can be accomplished for the arthritic by means of dieting. There is no necessity for the curtailment of red meat. I pointed out that among the numerous occupations there were only four employed as butchers. For some years I have been treating cases on a restricted diet, especially of carbohydrates. In the anæmic and atrophic cases a diet of red meats, fats, green vegetables and fruit is distinctly beneficial. Pemberton [5] has found that a safe and useful dietary can be drawn on a basis of 30 calories per kilo of body weight in persons who are of average body contour and not obese.

I have given no tables of results. Results in cases of arthritis depend on the nature of the case, the stage of the disease and the amount of amelioration we set out to obtain. Some cases we can cure, others we can relieve, while there are a great many for whom we can do little, as the sequelæ of the disease are worse than the disease itself.

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[1] JACCOUD, "Rheumatisme chronique fibreux de Jaccoud." [2] PONCET, *Lyon Méd.*, 1900, xciii, 91-228. [3] DOUTHWAITE, *Brit. Med. Journ.*, 1929 (i), 1035. [4] STRANGEWAYS (verbal conversation). [5] PEMBERTON, "Arthritis and Rheumatoid Conditions," Philadelphia, 1929.

TABLE I.

TOTAL NUMBER OF CASES (2,000).

	Hospital	Private	Total
Male ...	566	292	858
Female	659	483	1,142
			2,000

TABLE II.

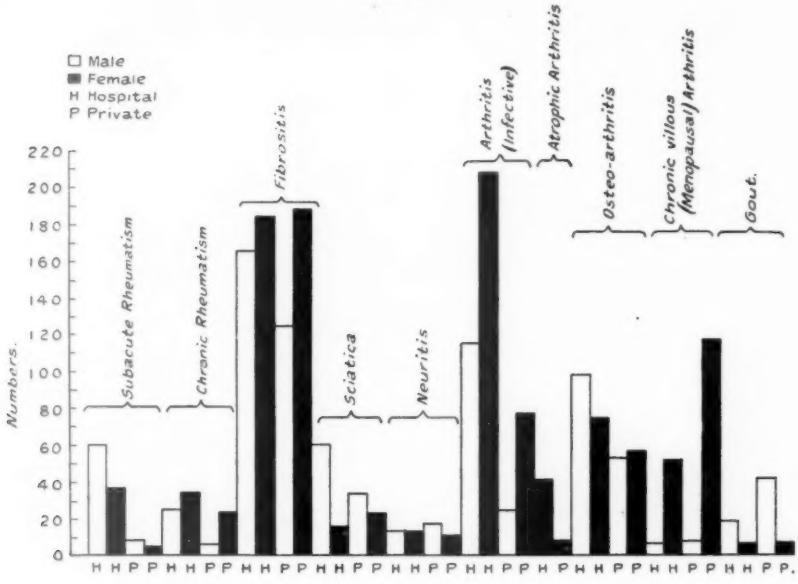

TABLE III.

Subacute Rheumatism. 111 Cases. M. 61, F. 47.

History of rheumatic fever ...	72
Endocarditis	39
Chorea	4
Enlarged thyroid	2
Family history	5
Tonsillitis	33
Dental sepsis	11
Erythema nodosum	2
Purpura	1
Psoriasis	1

Average age

Male	25·3
Female ...	25·06

Occupation. Males.	
Miners and outdoor workers ...	33
Indoor workers	15
Various	13

Occupation. Females.	
House duties ...	15
Textile trades ...	9
Shop assistants, etc.	7
School age ...	7

Chronic Rheumatism. 92 cases. M. 32, F. 60.

History of rheumatic fever ...	29
Endocarditis	10
Endocrine disturbance	7
Chorea	1
Family history	1
Tonsillitis	7
Dental sepsis	10
Iritis	1
Erythema nodosum	1
Shingles	1
Eczema	1

Average age.

Male	47·6
Female ...	48·4

Occupation. Males.	
Outdoor workers	15
Indoor workers	7

Occupation. Females.	
Housework ...	27
Textile ...	7

TABLE IV.

Fibrositis. 644 cases. M. 283, F. 361.

History of rheumatic fever ...	36
Family history	6
Enlarged thyroid	13
Artificial menopause	15
Dental sepsis	162
Tonsillitis	21
Bacilluria	5
Gallstones	7
Psoriasis	4
Erythema nodosum	5
Asthma	1
Eczema	1

Average age.

Hospital cases, M. ...	48·14
,, F. ...	44·3
Private cases, M. ...	50·6
,, F. ...	51·2
Total ...	48·6

Occupation.	*Males.*
Outdoor workers (miners, etc.) ...	98
Indoor workers	36
Various	9

Occupation.	*Females.*
Housework ...	112
Textile trade ...	21
Nurses, teachers, etc. ...	27

TABLE V.

Neuritis. 48 cases. M. 26, F. 22.

History of rheumatic fever ...	2
Oral sepsis	19
Tonsils	4
Colitis	1
Trauma	1

Average age.

Hospital, M. ...	51·5
F. ...	49·6
Private, M. ...	55·3
F. ...	45·5
Total ...	50·4

Occupations.	*Males.*
Outdoor workers	5
Indoor workers	6

Females.	
Housework ...	12

When definite fibrositic nodules have been found, cases have been included under fibrositis.

TABLE VI.

Sciatica. 134 Cases. M. 97, F. 37.

History of rheumatic fever ...	4
Dental sepsis	25
Tonsils	2
Colitis	1
Glycosuria	1
Gonococcus	1

Average age.

Hospital cases. M.	45·5
,, ,, F.	42
Private ,, M.	50·1
,, ,, F.	56·7
Total ...	47·85

Occupation.	*Males.*		*Females.*	
Outdoor workers: miners, engineering trade, labourers, etc.	42	...	Housework ...	12
Indoor workers	18	...	Various indoor ...	4

TABLE VII.

Proliferative Arthritis. 473 Cases. M. 148, F. 325.

History of rheumatic fever ...	33
Artificial menopause ...	10
Following pregnancy ...	14
Enlarged thyroid	14
Irregular menstruation ...	8
Endocrine disturbance ...	6
Dental sepsis	119
Tonsils	46
Bacilluria	11
Gonococcus	1
Leucorrhœa	17
Colitis	3
Psoriasis	6
Asthma...	5
Eczema...	2
Urticaria	3
Raynaud's disease ...	1

Average Age.

Hospital cases. M.	45·6
,, ,, F.	42·1
Private ,, M.	51
,, ,, F.	50·3
Total ...	47·2

Average age of onset, in 336 cases, 44·5

Occupation.	*Males.*
Outdoor:	
Miners, engineering, labourers, etc.	53
Indoor:	
Textile traders, shopkeepers, etc.	43
Various, and pensioners, etc. ..	25

Occupation.	*Females.*
Housework	163
School teachers and clerical ...	12
Textile	29
Shop assistants	8
Nurses	7
Various	19

TABLE VIII.—*Atrophic Arthritis.* **47 Cases.**

Said to have had rheumatic fever ...	6	*Average Age* ...	28·7
Leucorrhœa and abnormal menstruation	11	Average age commencement of disease	24·1
Condition followed on pregnancy ...	7		
Enlarged thyroid	7		
Dental sepsis	7	Married	21
Tonsils	7	Unmarried ...	26
Bacilluria	2		
Achlorhydria	2		
Asthma	1		
Occupations.		*Domicile*	
Housework 18		Large manufacturing towns ...	30
Textile 8			
Dressmaker 3			
School teacher ... 3			
Clerical 4			
Various indoor ... 2			

TABLE IX.

Osteo-arthritis. 279 cases. M. 149, F. 130.

History of rheumatic fever ...	20		
Dental sepsis	53		
Bacilluria	3	*Average age.*	
Colitis	1	Hospital cases, M. ...	56·3
Associated with trauma ...	39	,, F. ...	56·1
Static insults	11	Private cases, M. ...	59
Old history of T.B. joint ...	6	,, F. ...	57·2
Psoriasis	3	*Total* ... 57·1	
Eczema	3		
Leucorrhœa	1		
Tonsils	1		
Hip affected, male	56		
,, ,, female	27		
Spondylitis deformans ...	13		
Heberden's nodes	20		

Occupation. Males.		*Occupation. Females.*	
Engineering	17	Indoor, housework ...	55
Labourers, railway, miners, quarrymen	49	Textile	9
Joiners, plumbers	3	School teachers, clinical	4
Various, including pensioners ...	20	Various	8

TABLE X.

Chronic Villous Arthritis. 170 Cases. M. 10, F. 160.

Females.			
Menstrual history:			
Menopause	118		
Regular	7		
Irregular	9	*Average age.*	
Surgical menopause	11	Hospital cases ...	M. 51
No particulars	15	,, ,, ...	F. 53
Thyroid and endocrine	14	Private ,, ...	M. 59
Leucorrhœa	4	,, ,, ...	F. 57·25
History of rheumatic fever ...	4	*Total* ... 55	
Obese	7		
Trauma	11		
Flatfoot	35		
Bowleg	1		
Knock-knee	1		
Gallstones	2		
Heberden's nodes	31		
Colitis	2		
Migrane	1		
Urticaria	2		
Psoriasis	3		
Eczema	2		
Erythema nodosum	1		
Asthma	1		

Occupation. Males.		*Occupation. Females.*	
Outdoor	3	Housework ...	42
Indoor	2	Textile, dressmaker	6
		Various	5

TABLE XI.

Gout. 49 cases. M. 44, F. 5.

History of rheumatic fever ...	2
Dental sepsis	5
Bacilluria	1
Staph. alb. and *Strep. viridans*	1
Gonococcus	1

Average age.	
Hospital cases, M. ...	53·9
,, F. ...	60
Private cases, M. ...	55·1
,, F. ...	52·5
Total ...	55·3

Occupation.	*Males.*
Forgeman, smelterer, etc. ...	4
Railway	4
Manufactories	3
Miner	2
Tradesman	1

Occupation.	*Females.*
Housework ...	2
Domestic help ...	1

TABLE XII.

Incidence of Previous Attack of Rheumatic Fever.

Hospital cases.	M. 70 cases ...	59 definite attack, 21 with endocarditis 11 evidence of endocarditis, but no history of rheumatic fever
	F. 72 cases ...	61 definite attack, 23 with endocarditis 5 history doubtful 6 evidence of endocarditis, but no history of rheumatic fever 4 history of chorea
Private cases.	M. 30 cases ...	29 definite attack, 8 with endocarditis 1 evidence of endocarditis, but no history of rheumatic fever
	F. 28 cases ...	25 definite attack, 3 with endocarditis 3 history doubtful

Discussion.—Dr. W. EDGECOMBE said that among the sources of sepsis enumerated, it was surprising to find intestinal infection or intoxication only rarely appearing, in view of the fact that so many patients were sent to Spas for treatment for "rheumatic" diseases (after full investigation of all likely foci of infection), with the assumption, often made on insufficient grounds, that intoxication from the bowel was the underlying cause. Much of the vaccine treatment carried out on these lines was unscientific, and in practice quite useless. Might not the good results from cleansing of the alimentary tract by water and lavage be equally explicable on a "metabolic" as on an "infectious" hypothesis? Another point was the endocrine factor in causation. Here again we were apt to base our treatment on unproved theories. Much was said in a vague general way of "endocrine imbalance," but little was known with sufficient precision to form a basis for scientific treatment. Beyond the undoubted value of thyroid extract in the climacteric arthritis of women, and in certain other infrequent cases of arthritis, he questioned if any real benefit accrued in the "rheumatic" diseases from the administration by the mouth of other hormones such as ovarian, pituitary and suprarenal. In his hands such treatment had proved useless. Finally, one great disadvantage under which Spa physicians laboured was the difficulty of learning the end-results of their treatment, as they lost sight of the majority of their patients.

Dr. J. B. BURT said that he agreed that "atrophic arthritis" was a definite clinical entity, whether due to infection or to endocrine imbalance or to both. It was known under different names, such as "true rheumatoid arthritis," "infective," "indeterminate," and, on the Continent, as "primary chronic arthritis." He felt that the word "atrophic" was unsuitable. It was a pathological term and might be applied equally well to other types of arthritis in their later stages. The term "primary chronic arthritis" assumed no æticlogical factor, yet clearly distinguished the condition from arthritis secondary to focal infection, dysentery, pneumonia and gonorrhœa, these forms of arthritis being known as "secondary chronic arthritis." These two terms, "primary"—and "secondary—chronic arthritis," might be used with advantage in English nomenclature.

Dr. D. A. COLES said he hoped that the weight of this Section would be brought to bear on the Government in the matter of providing assistance towards the study and treatment of rheumatic troubles.

Dr. KING BROWN said that the Ministry of Health should encourage local authorities to undertake physiotherapy. The Metropolitan Borough of Bermondsey had decided to inaugurate such a centre, and the Ministry of Health had held an inquiry into the need of the district for its establishment.

Section of Dermatology.

[*November* 21, 1929.]

Pityriasis Lichenoides et Varioliformis Acuta.—A. C. ROXBURGH, M.D.

Cases of this disease have been shown here by Dr. MacCormac and Dr. Barber about 1922, by Dr. Gray in 1926 and 1928, Dr. Sibley in 1928, and by Dr. Haldin-Davis in January, 1929. This case of mine appears to be a typical one.

A. R., male, aged 15. Rash began as a few red spots on abdomen in first week of August, 1929. Patient came to St. Bartholomew's Hospital, August 20. Rash then involved whole trunk and upper arms; later spread downwards to wrists and down thighs and legs. Patient never felt ill; never had sore throat or any enlarged glands.

Family History: Nil ad rem.—Patient is a healthy looking lad, youngest of four children, all healthy. No tuberculosis in family.

Previous History.—Measles and whooping-cough only.

Present Condition.—Rash extends from top of head to soles of feet. Only a few lesions on scalp, but several maculopapules on both palms and soles. No lesions on mucous membrane of mouth.

Rash.—Consists of (*a*) pink macules; (*b*) papules, some of which are scaly and suggest psoriasis, while some are peeling from the centre outwards, like pityriasis rosea. Some are necrotic in centre and rather suggest varicella. When first seen by me in September some lesions appeared to be vesicular and some were like those in lichen planus. Patient thinks that papules are primary lesions, and that macules represent areas where papules have been. The rash does not itch. Wassermann and Sigma reactions negative (August and September, 1929). Pirquet test negative in November.

Blood-count (November, 1929).—Red cells 5,280,000, hæmoglobin 94%, colour-index 0·92. White cells 5,800 (*Differential*: Polymorphonuclears 72%, lymphocytes 23%, large mononuclears 4%, eosinophils, 1%).

Biopsy.—Two adjacent papules from back with normal skin at each end of section. At site of papules: Parakeratosis, elongation of papillæ, vacuolation of some prickle cells, dilatation of lymph spaces in papillary layer. Infiltration with small round cells in the papillary layer, and also around blood-vessels, sweat-glands, and arrectores pilorum muscles in the deeper layers.

Pityriasis Lichenoides et Varioliformis Acuta.—A. M. H. GRAY, C.B.E., M.D.

Patient, a girl, aged 5 years and 9 months, has had this rash for three weeks; the mother says it came on suddenly, and apparently does not itch. Lesions more or less confined to trunk, but extend somewhat on to limbs; almost same distribution as those of pityriasis rosea. Early lesions are faint pink, oval papules, which increase slightly in size and gradually darken. Milder lesions develop a mica-like scale, which eventually falls off. Lesions then flatten down, become pigmented macules and disappear. A certain number undergo necrosis. In some hæmorrhages occur, and lesions dry up as hæmorrhagic crusts, which, when they fall off, leave pitted scars. No vesicles are present.

This case is, in my opinion, one which links up those cases of necrotic lesions which have been described on the Continent under the name of pityriasis lichenoides et varioliformis acuta, with those described by Dr. MacCormac about six years ago.

Pityriasis Lichenoides et Varioliformis Acuta.—G. B. DOWLING, M.D.

Patient, male, aged 26.

Present Illness.—Eruption appeared first acutely October 4, 1929. It was very irritable and became more so in bed or in a warm room. Spots appeared first round wrist and thigh, spreading to shoulders and arms, and lastly to legs and feet. No spots have occurred on exposed places. The dry flat spots are the most irritable, and apparently cease to irritate after a scab has been formed.

On Examination: Eruption on trunk, arms, thighs, legs and feet, consisting of five types of lesion:—(1) Macular pink or yellowish-pink spots ranging in size from that of a large pin-head to that of a pea. (2) Slightly raised papules of about the same size. (3) Papules covered by a squama. (4) Papules with hæmorrhage in the centre, or hæmorrhagic scab. (5) Papules in healing stage, with depressed scar, from which a scab has become separated. It is evident that only a few of the lesions develop central hæmorrhagic necrosis.

Section, taken from a lesion from which a hæmorrhagic scab is separating, shows patchy cellular infiltration throughout the dermis, extending down to the level of the sweat-glands, and somewhat densely aggregated around them, elsewhere for the most part around vessels. Polymorphonuclear leucocytes are found in numbers in the infiltrate. A hæmorrhagic scab can be seen over a regenerating epidermis.

Pityriasis Lichenoides et Varioliformis Acuta.—J. E. M. WIGLEY, M.B.

Patient, male, aged 24, healthy. Eruption first noticed about six weeks ago, following a scratch on the wrist. No itching or other sensation noticed. Lesions distributed over trunk, arms and upper portion of the thighs; most copious on forearms. They consist of small scaly papules, small blood-crusted macules and purpuric spots. Wassermann reaction, negative. The case was seen for the first time yesterday, and is shown to compare and contrast with the two cases just described.

Discussion.—Dr. H. MACCORMAC said that a number of these cases had come under his observation in 1921. Two of them he had exhibited at a meeting of the Section.[1] The eruption was in many respects similar to that presented by the patients now exhibited, except that in none of them was any necrosis observed, nor was there any scar formation. The eruption had cleared up completely in two of the cases, after being present for about four to six months, a feature which distinguished it from the accepted form of parapsoriasis.

Dr. HALDIN-DAVIS said that he might have added another case to to-day's series, one which he showed a year ago. He had seen the patient only the previous day and the condition had remained almost unchanged although the varioliform lesions seemed to lose their virulence as time went on. In his case, that of a youth, the old scars were still visible, but there were no more large necrotic papules occurring.

Dr. H. W. BARBER said he was glad to hear Dr. Gray's opinion that these cases were identical with those shown by Dr. MacCormac some years ago, but he believed that there were no necrotic lesions in Dr. MacCormac's cases. In most of those which he had personally observed, the eruption had come out acutely within a few days, like an acute psoriasis or pityriasis rosea. In the case of one patient, who also had lupus erythematosus, injections of krysolgan had, to his surprise, cleared up the eruption, and he had recently heard that another patient had also recovered after two years. In this case, too, he had advised injections of krysolgan.

1 *Proc. Roy. Soc. Med.*, 1923, xvi (Sect. Derm.), 62.

Dr. J. A. DRAKE said that as a group of these cases was being seen, it would be interesting to know whether they showed any time-incidence, i.e., whether they appeared particularly in spring or autumn. Most of them showed the peculiar stream-like distribution along the lines of the ribs and in the axillary folds, and the greasy mica-like scale was also like that seen in pityriasis rosea. Hence it was possible that the two diseases had a similar cause.

Dr. S. E. DORE (President) said that one of Dr. Gray's cases had come under his observation. In that case the necrosis had been strongly marked and there had been large, oval, stippled scars.

Dr. A. M. H. GRAY (in reply) said that the reporting of a series of cases in this way threw light on a feature one wanted to know about, i.e., the duration. When Dr. MacCormac had shown his first case, it was a question whether it was one of pityriasis lichenoides chronica (*parapsoriasis en goutte*). Against this view was the fact that some of these cases lasted only a short time. When Mucha first described this varioliform type about thirteen years ago, he and those who saw the case with him were struck with the resemblance of the early lesions to those of pityriasis lichenoides chronica, and thus the present cumbersome name was attached to the disease. It now appeared that this was a more persistent complaint than had been thought. It was almost impossible to distinguish the two conditions clinically; in the cases now shown the condition might prove to be an acute necrotic form of pityriasis lichenoides chronica.

Unusual Form of Sarcoid (?). Case for Diagnosis.—H. W. BARBER, M.B.

M. M., female, aged 47, married. Lesion on right leg began about six years ago as a "bruise," and later patient was "stung" over same area. No history of serious illness.

On the anterior surface of the right leg is an indurated area about two inches long and whitish in colour, surrounded by a deep lilac-coloured border, spreading out from which, over the central white portion, are arborescences of dilated vessels. In the central white area are three small circular depressions, which have apparently arisen spontaneously.

The waxy indurated central portion suggests scleroderma, but the case resembles others that have been shown by, I think, Dr. Gray and Dr. Goldsmith, and Dr. Dowling. I believe the lesion in the case shown by Dr. Gray and Dr. Goldsmith was proved to have a tuberculoid structure.

Unfortunately I have not been able to make a biopsy in this case, but I am inclined to negative the diagnosis of scleroderma, and to suggest that the condition may be a form of sarcoid.

Several injections of solganal have produced a slight improvement.

The patient also has xanthoma palpebrarum.

Discussion.—Dr. H. MACCORMAC said that Dr. J. H. Stowers and himself had each shown a patient with an identical eruption, at a meeting of this Section in October, 1921.[1] In his own case the lesions on the leg were accompanied by a hypodermic nodule in the right thigh. He had then suggested that the condition was a species of tuberculide, and this view seemed to be confirmed by Dr. Goldsmith's investigations in a similar case exhibited at a meeting of the Section in June, 1928.[2]

Dr. DOWLING said he had exhibited a similar case about a year ago.[3] The patient was a woman of middle age, who had had an oval lesion in the same position and of about the same size, for a number of years. Side by side, the two cases would have appeared identical. The histological structure of the edge was definitely tuberculous, consisting of a dense subepidermal infiltrate composed mainly of epithelioid cells with some giant cells.

The case had been shown as an unusual type of sarcoid.

(?) Lupus Erythematosus of the Buccal Mucous Membrane.—H. W. BARBER, M.B.

J. G., female, aged 41, married. Six children, three living; no miscarriages. The condition of the tongue has been present for about two years. I believe it was first erroneously diagnosed as specific: at any rate, in spite of a negative Wassermann reaction, antispecific treatment and injections of neosalvarsan were given, without

1 *Proc. Roy. Soc. Med.*, 1921-22, xv (Sect. Derm.).
2 *Ibid.*, 1928, xxi, 1768 (Sect. Derm., 80).
3 *Ibid.*, 1765 (Sect. Derm., 77).

benefit. The tongue and the inner surfaces of the cheeks are involved. The dorsal and lateral surfaces of the tongue are covered with irregular white patches: apart from these there appears to be definite inflammatory reddening, with a certain degree of atrophy: on the left side there is, or was, a small ulcer. On the mucous membrane of the cheeks are white patches; careful examination shows that these are situated on an erythematous base.

At first sight, one would, I think, diagnose lichen planus, but against this, in my opinion, are the reddening and atrophy of the tongue, and the erythematous patches on the cheeks. There are no lesions on the skin. At my request Dr. Forman performed (1) the von Pirquet test, using human and bovine tuberculin: this was completely negative in all dilutions, as is shown in the accompanying photograph; (2) intradermal tests with emulsions of (*a*) *Streptococcus hæmolyticus*, (*b*) *S. viridans*, (*c*) staphylococci, (*d*) normal saline. (*a*) was very strongly positive, (*b*) rather less so, (*c*) slightly positive.

There is severe oral sepsis which is under treatment. My diagnosis is lupus erythematosus of streptococcal origin.

Discussion.—Dr. A. M. H. GRAY asked whether Dr. Barber had seen or read of lupus erythematosus on the mucous membrane having been diagnosed in the absence of lesions on the skin. He appreciated the points which Dr. Barber raised in this case, but he did not think sufficient evidence had been produced in favour of his contention. He was not convinced that the condition was not lichen planus.

Dr. GRAHAM LITTLE said he regarded this case as almost exactly like the one he himself was showing, and he was glad of the suggested diagnosis.

Dr. BARBER, in reply to Dr. Gray, said he had seen one case in which he had diagnosed lupus erythematosus of the buccal mucous membrane without cutaneous lesions. About a year later the eruption appeared on the cheeks. He had shown the present case with a query as to the diagnosis between lupus erythematosus and lichen planus, but he thought the former was the correct one.

Extensive Lichen Planus with Involvement of the Mucous Membranes in a Young Child.—H. W. BARBER, M.B.

T. C., male, aged 3 years and 8 months. There is an extensive eruption of lichen planus, which began on the ankles. The tongue, mucous membrane of the cheeks, and the vermilion borders of the lips are involved.

The case is shown owing to the rarity of such extensive lichen planus in a young child.

I have recently had an equally extensive case in a child aged 6, and in that case the confluent patches closely resembled psoriasis.

Discussion.—Dr. J. E. M. WIGLEY said that six weeks previously he had had a case of lichen planus in a baby aged 2½ years, who had a line of typical plain papules extending down the outer side of the left arm and the back of the left leg. At the same time there was an area of papules round the upper abdomen, corresponding to the tenth dorsal segment. When he saw the child the eruption had been there a month; it was said not to itch. The child was otherwise healthy. The lesion not present at birth. It had disappeared at the end of three weeks.

Dr. J. M. H. MACLEOD said he had had a case in a young child and had thought that the condition was lichen planus, but it had proved to be nævus unius lateralis. His earliest case of lichen planus was in a child aged 4 years.

Dr. GRAHAM LITTLE asked whether it was not the general experience that linear lichen planus was more common in children than was lichen planus of the ordinary type. He was doubtful about the condition in the linear cases being true lichen planus. Some of the cases shown as such were probably cases of linear nævus.

Dr. A. M. H. GRAY said he had shown a case in a child under 3 months old, and there had been some difference of opinion concerning it. The lesion was not a nævus, because it came and went while under observation. The suggestion was made at the meeting that it was lichen urticatus, but he did not accept that. He had seen two other cases of the same kind in children under a year old, and he regarded the condition as genuine lichen planus.

The PRESIDENT said he had recently seen two cases of apparent multiple linear warts, in which the condition had proved to be lichen planus. This was a possibility to be considered in the case of children.

Norwegian Scabies.—G. B. DOWLING, M.D.

I. K., female, aged 29.

History.—About nine months ago first noticed that toe-nails were becoming thickened. Ultimately, thickness increased to a degree which made walking in shoes almost impossible. Changes in nails were accompanied by horny thickening on undersurface of metatarso-phalangeal joints, and mildly irritable papular eruption between toes and on adjacent portion of dorsum of feet. Similar eruption appeared in interdigital spaces of hands and about wrists, and a few papules were present on forearms and on legs. Upper arms, thighs, and trunk were not attacked.

The patient was seen by me at the West London Hospital for the first time about six weeks ago. All the toe-nails were thickened and crumbling. Hyperkeratotic masses were also present on the ball of each foot. A piece of friable toe-nail was removed for examination for fungus, with negative result. All the toe-nails were then removed and sent to the laboratory for further examination. Dr. Elworthy reported an infection of the nails and epidermis with a member of the family Sarcoptidæ. He kindly prepared microscopical specimens which, to quote his description, "show adults with some pregnant females, nymphs, developing ova, and empty shells occurring in variable numbers, usually large, in different parts of the diseased nails and cuticle." We intend, with the material at our disposal, and with the help of an entomologist, to find out whether the acarus present is identical with that of ordinary scabies.

The PRESIDENT said he did not think that a case of the kind had been shown to the Section before. It was generally stated that the disease was due to a sarcoptes derived from the wolf, but in this patient there was no history of infection.

Hidradénomes éruptifs of Darier and Jacquet.—W. N. GOLDSMITH, M.D.

S. G., male, aged 22.

History.—The nodules under consideration are said by the patient to have been present as long as he can remember; he thinks since birth. They do not trouble him, and he consulted me not about them, but about a rash on the face and a carbuncle on the back of the neck. These cleared up rapidly, but have since recurred.

On Examination: August, 1929.—On the chest and abdomen are numerous pale yellow or mauve cutaneous nodules about the size of a pea; overlying epidermis is unchanged.

I considered the possibilities of xanthoma, *hidradénomes éruptifs* or, conceivably, urticaria pigmentosa. Against xanthoma was the rather mauve colour, the even size of the papules, and the absence of any evolution. Against urticaria pigmentosa was the absence of any marked pigment or any urticarial reaction on rubbing, and again, the uniformity in size and degree of elevation of the individual nodules.

Histological examination proved the condition to be hidradenoma. This variety affecting the chest and abdomen is very rare. It is far more common in women, and generally makes its appearance about the age of puberty. On this account it has been thought that the little tumours arise from apocrine sweat-glands, which are

more numerous and active in women, and only become fully developed at puberty. This idea never seemed to me probable, since the distribution does not correspond with the main location of the apocrine glands, namely, axillæ, nipples and pubes. It is, therefore, of peculiar interest to find this eruption occurring in a male at an extremely early age.

Dr. J. M. H. MACLEOD asked whether any Member could say what was the earliest age at which this condition erupted. He thought such cases began very early in life. He regarded the lesions as nævi.

Giant Granuloma Annulare (?).—W. N. GOLDSMITH, M.D.

E. B., male, aged 80, an actor.

History.—Duration about three months. He first noticed a swelling on the back of the right arm, which slowly spread. A month later one appeared on the back of the left leg. When seen about a month ago there was a wavy circle at the back of his right arm, consisting of a steeply raised smooth ridge, slightly mauve in colour and of a rubber-like consistency. It was about half a centimetre in height. The portion of skin enclosed by this wall (which was interrupted at a few points) appeared normal. No subjective symptoms.

Present Condition.—The rest of the lesion at the back of the right arm has flattened down considerably, but there is on the back of the left leg an oval, purplish-red, firm tumour of similar consistency, but deeper in colour than the original lesions. A little further down the leg, more or less concentric to the large tumour, the newest lesions are to be seen, being elongated infiltrations forming part of incomplete circles.

Wassermann reaction negative.

Histology.—Massive inflammatory exudate, chiefly of lymphocytes.

Clinically this case is closely similar to that shown by Dr. Gray, on June 16, 1927.[1] In that case there was a centrifugal spread, and the skin which had been affected showed a parchment-like change. A histological section revealed large numbers of giant cells, rather peculiarly arranged, and not forming part of tubercle systems. It may be that such an appearance represents a later phase in the evolution of a lesion than is illustrated by the section from my case. Dr. Gray's patient was completely cured by a few doses of X-rays. The complete absence of itching makes a diagnosis of mycosis fungoides unlikely. Professor Jadassohn, who saw Dr. Gray's patient and the histological section, suggested that it might be a sarcoid of the Darier-Roussy type.

Discussion.—Dr. H. W. BARBER asked whether Dr. Goldsmith thought that the large purple tumour was of the same nature as the granuloma annulare lesions on the arm and leg.

Dr. MACCORMAC said that if one sought for a single diagnosis embracing all the phenomena, such conditions as mycosis fungoides or an unusual leukæmic eruption might be considered. The absence of itching was against both; he was inclined to regard the process as a tuberculide of the sarcoid type.

Dr. GOLDSMITH (in reply) said he considered that the purple tumour was of the same nature as the elongated lesions. The patient's blood-count was normal.

Subacute Generalized Exfoliative Dermatitis of Wilson-Brocq.—W. N. GOLDSMITH, M.D.

M. R., a girl, aged 7.

History.—Duration six months. At end of May, 1929, patient fell and cut her right knee. A fortnight later red sores and blisters appeared around the cut and then a red crusty rash spread over both legs and arms. First seen by me July 26, 1929,

[1] *Proc. Roy. Soc. Med.*, 1927, xx, 1834 (Sect. Derm., 114).

when I noted "eyelids crusted, arms and legs red and rough and crusted; here and there flat impetigo-like blisters." I made the diagnosis of staphylo-seborrhœic dermatitis. Treatment with 1% monsol in linimentum calaminæ was prescribed.

Admitted to hospital, August 9, 1929.

On Examination.—Head and neck: Whole face and scalp reddened except round the mouth; irregular white scales; no moisture. Chest: Smaller irregular red patches with rather yellower impetigo-like crusts. Back and abdomen: Uniformly red and rough, but only minimal scaling. Arms: More intensely reddened and scaly, but hands practically free. Legs: More involved on anterior aspect; distal half of feet free.

General health very good. Temperature generally between 99° and 100° F. in the evening. For the next four months the condition was unchanged. The whole body surface was involved, except some islands of perfectly healthy skin on the upper part of the chest, palms, soles and dorsal surface of hands and feet. Rest in bed, calcium internally, weak sulphur and salicylic ointment, ultra-violet light and, more recently, arsenic, produced no perceptible change for better or worse, except that scaling was naturally reduced and the child made much more comfortable by the ointment. No subjective symptoms. The reddened skin was distinctly infiltrated and, over the wrists and insteps, was lichenified. There was never any distinct follicular arrangement.

The temperature became normal and, suddenly, a few days ago, a great improvement took place. The skin became smoother and more mauve in colour. No change in treatment had been made, but possibly the arsenic had accumulated to an effective concentration. Blood-count normal.

Diagnosis.—My original idea of seborrhœic dermatitis was negatived by the site of onset, almost universal distribution, absence of subjective symptoms, and complete lack of response to treatment. Pityriasis rubra pilaris must be excluded by the absence of any follicular lesions. One cannot class it as a secondary erythrodermia, in spite of the presence of a few impetigo-like lesions at the beginning. They cleared up very rapidly and were probably due to scratching, caused by a little initial pruritus. Hebra's pityriasis rubra cannot be diagnosed with certainty until the contracted atrophic stage has set in. There are usually general symptoms. This case seems to correspond most nearly to those of the rare condition known as subacute generalized exfoliative dermatitis of Wilson-Brocq. This has an average duration of from three to eight months. Most of the cases described have been in adult men. It begins, as it did in my case, at a single point, generally at a joint, and spreads to the whole body, involving the hands and feet last. The generalization is very rapid.

The only point which makes me doubtful is that Brocq laid stress on the complete loss of hair and nails, or a severe dystrophy early in the disease. In this case there was some falling of hair, but it was not striking, and the nails are not very dystrophic, though the condition is now six months old.

Discussion.—Dr. DOUGLAS HEATH said he considered this to be a case of erythematous psoriasis. Dr. Adamson had published a series of cases of pityriasis rubra pilaris in children and some of them subsequently developed psoriasis, so evidently there was a close relationship between the two diseases. Often one could see an exactly comparable condition to this on the face in a case of generalized psoriasis. It would be interesting to know if there was a history of psoriasis in this patient's family.

Lichen Scrofulosorum.—R. T. BRAIN, M.D. (for Dr. W. J. O'DONOVAN).

T. N., an unmarried ex-soldier, aged 19, first attended the skin department of the London Hospital in October, 1929. Skin trouble had begun about three years before, as eruption of pimples across lower part of back and on limbs. Lesions soon became universal. There was no itching. At this time patient was in the Army, and was treated at several hospitals without apparent benefit. Eighteen months

ago was admitted to hospital at Greenwich, and at one period was treated by irradiation with carbon arc lamp. Following this treatment "red lumps" appeared on his chest. Three weeks after he was discharged from hospital coarse scales appeared on upper part of trunk and on forearms and hands.

He was admitted to the Royal Free Hospital where he was seen by Dr. Haldin-Davis who showed him at a meeting of the Section in October, 1928, as a case of lichen plano-pilaris.[1] After investigation he was shown again in December, 1928, as a case of ? benign lymphogranulomatosis.[2]

Case shown by Dr. R. T. Brain for Dr. W. J. O'Donovan.
Lichen Scrofulosorum.

He was treated for a considerable time with light baths and the infiltration diminished greatly. Sanocrysin was also given but without appreciable benefit and eventually he was transferred to an infirmary.

Previous health good; no cases of tuberculosis known in his family.

1 *Proc. Roy. Soc. Med.*, 1928, xxii, 92 (Sect. Derm., 4).
2 *Proc. Roy. Soc. Med.*, 1929, xxii, 501 (Sect. Derm., 13).

On Admission.—General health good. Lymphatic glands in neck, axillæ and groins showed moderate enlargement; they were discrete and firm in consistency; overlying skin was normal in colour.

Much of the skin was normal in texture and appearance, but on upper part of trunk and on proximal segments of limbs there were patches of coarsely roughened skin with a nutmeg-grater-like surface.

The individual skin lesion consisted of an enlarged conical follicle, from the apex of which projected a horny plug surmounted by a silvery scale and readily expressed or removed by forceps, leaving a pink, moist, vascular crater. Examined in liquor potassæ under the microscope, it was found to consist of horny scales and granular material surrounding a fine hair. The base was of a pale pink colour, but there was no palpable infiltration of the skin. Aggregations of these prominent follicles formed large irregular sheets almost covering the dorsal region of the back. On the chest the eruption was more patchy, whilst the upper arms and thighs were much less affected. Two pieces of skin were removed for histological examination and each presented a typical picture of miliary tuberculosis. There was considerable infiltration around the hair follicles, many of which were grossly distended into the shape of goblets full of keratin. Early miliary tubercles could be recognized in the walls of the follicles. The infiltration with spindle, stellate and round cells was also well marked in the vessel-walls, and extended as uniform sleeves along the capillaries to the interpapillary processes where they frequently terminated in a miliary tubercle.

In addition to the lesions of the skin there were definite nodules and tumours of the hypodermis. These consisted of firm well-defined, rubber-like masses lying between the skin and the deep fascia, varying in size from minute nodules to ovoid tumours 3 × 2 cm. They were found on the hands, forearms and shins, and over the upper lumbar spines.

Microscopic examination revealed a gummatous structure with miliary tubercles in the periphery of the section. There was less caseation than is usually seen in tuberculous nodules of this size.

No other evidences of tuberculosis could be found, apart from the lesions in the skin, in the hypodermis, and in the lymphatic glands.

The heart and lungs were sound and the negative findings in the chest were confirmed by X-ray examination. The blood-count was normal. The urine was pathological only in containing a few leucocytes.

Wassermann reaction negative. Von Pirquet reaction weakly positive. An injection of old tuberculin produced no general reaction.

Guineapig inoculation has been carried out, but although more than three weeks have elapsed, the animal continues to flourish and to gain in weight.

Discussion.—Dr. HALDIN-DAVIS said that the patient's condition had altered a good deal in the year since he came under his (the speaker's) care. There had then been a jelly-like infiltration of the skin, but the skin was now almost normal in texture, except for the numerous and prominent spines, which had not been present before. Then the glands had been much enlarged, masses of them being both visible and palpable behind each lower jaw. Before investigation it had been suggested that this glandular enlargement was due to syphilis, but that had been disproved, and there was no doubt that the condition bore a much greater resemblance to tuberculosis.

He regarded it as a tuberculide. At one time he thought it was benign lymphogranulomatosis. The man seemed now to be better in health than he had been a year previously.

Granuloma Annulare.—E. G. GRAHAM LITTLE, M.D.

The patient, a girl, aged 4½ years, has a lesion typical of the disease, covering an area of 2 in. diameter on the dorsum of the right foot; immediately above this patch is a smaller and rather redder raised patch about the size of a sixpence. No subjective symptoms; lesions have grown slowly by peripheral extension. The child seems otherwise well. There is an interesting history of a maternal aunt at present suffering from Bazin's disease, under the care of Dr. Darier in Paris.

Folliculitis Decalvans et Atrophicans.—E. G. GRAHAM LITTLE, M.D.

The complex of symptoms shown in this case has become recognized under the above title since I showed the first case of it in 1915 at a meeting of the Section.[1] A second case was shown about two months later by Dr. Dore[2]; three or four months later Dr. Wallace Beatty wrote an interesting paper, in which he contributed a third case of the same complex, and said that he was inclined to regard the condition as an example of Darier's disease.[3] Since then a number of other cases have been recorded, and I myself have had opportunities of seeing this complex in combination with undoubted lichen planus, and have recorded the opinion that it is really a variety of lichen planus, hitherto undescribed in textbooks. The present case is a further confirmation of this view. The patient, a single woman, aged 52 has been under observation by her doctor for about two years. She first consulted him on account of patches of follicular eruption about the lower abdomen and the trunk and back; these were almost certainly patches of lichen spinulosus. The scalp became affected only about twelve months after the eruption appeared on the body. At present the scalp alone is affected. There are areas of cicatricial alopecia scattered over it with some still active inflammation of the hair follicles.

In the pubic area, in the positions where the lichen spinulosus had been observed, there is a widespread loss of hair. On the buccal mucosa of the cheeks there are patches of typical lichen planus, but no lichen planus can now be seen anywhere else, nor is there any lichen spinulosus now present.

Discussion.—The PRESIDENT said he agreed that the patch on the mucous membrane of the mouth was typical lichen planus, and he thought the exhibitor had established a new syndrome, or a new type of lichen planus.

Dr. BARBER said that these cases were of great interest and importance, and their significance was not appreciated abroad. He had seen, with Dr. Dowling, a girl who had cicatricial alopecia of the scalp and lichen spinulosus of the body: typical lichen planus had then developed. He had shown a man last year who had a patch of cicatricial alopecia, lichen spinulosus, and lichen planus, coexistent at the same time. He suggested that the so-called "pseudo-pelade" was really lichen planus of the scalp, and that liche spinulosus in adults was usually the follicular type of the same eruption.

Dr. WIGLEY said he had had a case of perfectly typical lichen planus. When lesions which were on the arms and legs had faded, spiny horny lesions developed on the forearms three inches below the elbows and these appeared to be typical of lichen spinulosus.

Lichen Planus (?) Lupus Erythematosus (?). Case for Diagnosis.—E. G. GRAHAM LITTLE, M.D.

I am showing this case in order to obtain suggestions as to treatment. There is a remarkable history. The patient, a woman, aged 44, has been under her doctor's care for the past two years. In May, 1928, she had a painful superficial ulceration in the mouth, near the left commissure, which had persisted for some months. There was a similar but smaller lesion near the right commissure. The condition was regarded as a superficial leucoplakia, and friction from her dentures was suspected. These were altered, and treatment by ultra-violet light and X-rays was carried out during the same month. She appears to have had nine doses of X-rays, followed later by five doses of ultra-violet light. In June, 1928, she was sent to the Radium Institute. Dr. Hayward Pinch, the Medical Superintendent, regarded the lesion on the left side of the mouth as "a patch of leucoplakia of the second degree," and gave her five exposures of radium of 1½ hours' duration at intervals of some weeks with a suitably sized full strength applicator, screened with $\frac{1}{10}$ mm. of lead.

1 *Proc. Roy. Soc. Med.*, 1915, viii (Sect. Derm., 139).
2 *Ibid.* (Sect. Derm., 247).
3 *Brit. Journ. Derm.*, 1915, xxvii, 331.

The condition appears to have improved for a certain time after the radium treatment, but has now recurred as badly as ever. The patient had been seen recently by Sir Crisp English, who advised the surgical removal of the patch. It was in these circumstances that she was sent to me.

The condition is obviously the same as that in the case shown at this meeting by Dr. Barber with a query diagnosis of lupus erythematosus. The present appearance is that of a superficial white patch upon a red base. There is no ulceration now visible, and in view of the numerous applications of radiation of various kinds, it is difficult to estimate how much of the present appearance is due to the treatment, as distinguished from the disease. The patient gives a somewhat dubious history of a small red patch on the left cheek near the ear, which persisted for six months, but which has completely disappeared without leaving any atrophy. There is no history of any other skin eruption. Were it not for the history of ulceration, I should be inclined to make a diagnosis of lichen planus, but I have never seen ulceration following upon lichen planus of the mucosa.

Dr. BARBER said that the patient should not have any further applications of X-rays or radium, nor would he contemplate excision of the patch in its present condition. He thought lupus erythematosus a possible diagnosis, especially in view of the history of a patch on the cheek. He would try the effect of gold compounds.

Necrotic Tuberculides (?) and Granuloma of Sarcoid Type: Case for Diagnosis.—H. C. SEMON, M.D.

J. H., male, aged 56, builder's labourer.

History.—(Abstract of notes by Ministry of Pensions.) Enlisted A1, August 28, 1915. No history of previous disease. Impetigo, June 1917; impetigo, July 1918. Invalided from Egypt, January, 1919. Sycosis, General Hospital, Birmingham. Boarded January 1, 1919, on account of sycosis of chin and cheeks. Beard affected later, has been epilated twice. May 11, 1920: Rash on cheeks and temple. A few sores on neck. Both ears "eczematous," sores on left hand. July 12, 1921: Septic sores on face and beard region, ears and left hand. A case of recurrent sycosis. First seen by me in March, 1922. Several small callous circular ulcers on knuckles and back of hands. Definite ulceration of tip of left concha and numerous scars of previous lesions on back of hands, right side of neck, and bridge of nose. Wassermann reaction was negative. Patient was lost sight of for five years, and attended two London hospitals, also Ministry of Pensions hospital at Sidcup, where light treatment was given with benefit. He came to the Royal Northern Hospital on November 11, 1929.

Present Condition.—Scars of old ulceration as above indicated. Callous ulcers, varying in size from $\frac{1}{8}$ to $\frac{1}{4}$ in. in diameter on knuckles and backs of hands. Similar ulcers on both pinnæ. Whole of left upper lid is occupied by a soft granulomatous non-ulcerated tumour which in no way inhibits the movement of the eye, and is quite painless (present, 2 years). The uvula is covered by a membranous deposit, which however, has not produced either pain or discomfort.

The investigations are not completed to date. They include: (1) Culture of epilated hairs, on Sabouraud's medium, one week: negative. (2) Mantoux's T.B. test: negative, 1/10,000; positive, 1/1,000. (3) Biopsy from granuloma of lid reveals a normal epidermis immediately adjoining which are packed masses of lymphoid cells, which in places resemble those of a round-celled sarcoma. There are no giant cell systems, and there is no tendency to necrosis. There has as yet been no opportunity for a detailed examination of the chest with radiograms, etc.

Suggested Diagnosis.—Ulcerative necrotic tuberculides on the ears, beard region and knuckles. Granuloma of "sarcoid" type on left upper lid. The possibility of artefacts, as suggested by one of the Members, regarding the hand and ear lesions, has to be taken into serious account, but it does not explain the granuloma of the eyelid which is of obvious neoplastic origin.

Discussion.—Dr. SYDNEY THOMSON regarded this as a typical cutaneous leishmaniasis so far as the lesions on the hands were concerned. When Delhi sores occurred on the face they gave rise to curious sycotic conditions, also to very variable nodular conditions. He had known cutaneous leishmaniasis to last ten years and then clear up only under treatment with antimony.

Dr. SEMON, in reply, said that he had had many opportunities of studying Delhi sore. In his experience, although the lesions in that condition might take as long as a year to heal, new ones did not continue to make their appearance for any length of time after the patient had left the East. He had never seen a neoplastic manifestation, such as that in the present case, in connection with the disease. In all his cases the clinical appearances were those of an ulcer.

POSTSCRIPT.—A microscopic examination for Leishman-Donovan bodies has since been carried out with negative results.—H. S.

War Section.

President—Group Captain Hardy V. Wells, C.B.E., R.A.F.

[*December* 9, 1929.]

Some Remarks on the Evolution of Naval Hygiene.

By A. E. Malone (Surgeon Commander, R.N.).

This paper is not an attempt to teach the principles of ship-building, but an effort to describe briefly a few of the hygienic problems which have arisen in the course of the evolution of the modern ship.

During this period great changes have taken place. The rafts and canoes of primitive races have been succeeded by oars and sail, and open boats have been decked in: sail in its turn has given way to steam, and iron has replaced wood. An increase in size and encroachment on internal space has marked each stage in this transition. The advent of the turbine and internal combustion engines, of electricity and of oil fuel, has also been epoch-making in naval affairs. Apart from other changes they brought about, they all opened up new chapters in marine sanitation. The first difficulty probably occurred when the frames of the open boat were boarded over to make a smooth deck to the vessel, thus forming a hidden space or bilge where water and filth could collect. The rivalry between Rome and Carthage saw the construction of large fighting galleys, which were often completely decked over. Primarily built for fighting, like our warships to-day, they, too, must also have introduced some problems in ship ventilation.

The re-discovery of the mariner's compass at the beginning of the fourteenth century opened up a vista of ocean navigation and stimulated ship construction. Ships gradually became bigger and stronger, decks were added, and they began to be used as permanent habitations. In the earlier decked ships hatches were the only means by which air could get below, for it was not till about 1500 that portholes were introduced by a French shipbuilder at Brest.

In addition to being very inadequately ventilated, ships were overcrowded and damp. What air managed to get below was vitiated by human agencies, by the live stock which was then frequently carried to provide fresh meat, by foul odours from the bilges, or by the combustion of the candles used for lighting purposes. The crews were generally poorly clad and frequently without a change of clothing, while the restricted water supply and absence of soap curtailed the opportunities for washing. In the reign of Charles I it was complained "that the men stink as they go and their poor rags are so rotten that they are ready to fall off."

It is little wonder then that the great voyages of discovery which began in England under the Tudors, furnish us with accounts of sickness and disease, squalid accommodation and rotten food. For nearly 300 years scurvy was the curse of the sea and it is estimated that over 5,000 deaths took place annually among our seamen from this disease alone.

Its final elimination towards the end of the eighteenth century is one of the first triumphs of preventive medicine and was largely due to the work of a naval surgeon, Dr. Lind, who, in 1753, published a treatise on scurvy. Fifteen years later Captain Cook put Lind's teaching into practice and kept his ships free from scurvy, a thing that had never been done before. Cook's hygiene was considerably in advance of his time and embodied many of the principles we still practise. Constant vigilance, daily personal inspection of his ship, cleanliness, dryness and green food whenever possible, were some of his maxims.

To-day scurvy is a rare disease on shipboard and the only reminder we have of it in the Navy is the lime juice issue, which was first ordered by the Admiralty in 1795, chiefly at the instigation of Gilbert Blaine, a disciple of Lind's. The monthly issue of soap on repayment has also come down to us from Blaine's time.

In addition to his treatise on scurvy, Lind (1716-99) drew up rules for ship-hygiene and the prevention of typhus, then a prevalent disease in the Navy, owing to the custom of manning ships with the contents of the jails. Bad as the conditions in jails were, those in ships were worse, for, as Dr. Johnson remarked, "no man will be a sailor who has contrivance enough to get himself into jail; for being in a ship is being in jail with a chance of being drowned." Lind, however, persuaded the Admiralty to establish "slop ships" where men were segregated and cleansed before being drafted to sea-going ships, and succeeded in reducing the incidence of typhus afloat. He also introduced into ships a small "still" for making fresh water, the supply of which was always a source of trouble. The records of the early navigators furnish us with little information about fresh water between their home port and the first land touched at, but contemporary accounts indicate that their supplies cannot have been above suspicion.

Water was carried in barrels in the hold of the ship, or in casks on deck, and was replenished as occasions offered, either by spreading sails and catching the rainfall, or, if near land, by sending boats ashore to bring off fresh supplies.

Complaints about drinking water, especially as regards its smell and taste, were common, and it was frequently blamed for causing disease. The foul smell which so often developed in the casks on deck was probably due to some kind of algæ, as there never seem to have been complaints about the barrels in the hold, where the daylight necessary for the development of this form of life could not penetrate.

A similar state of affairs is sometimes experienced in reservoirs to-day, where the nauseous odour and taste caused by the bursting of the algal oil sacs may make the water quite undrinkable for a few days.

Anson, in his "Voyage round the World" in 1740, gives a good account of such an occurrence in the water obtained in the island of St. Catherine off Brazil.

In 1772 the Admiralty ordered Lind's still to be fitted in all warships, but this instrument was only capable of making very small quantities of water. About forty years later two-ton iron tanks were introduced instead of casks for storing water, and we may regard these as the forerunners of the permanent compartments, the fresh water tanks, which are to-day built into every ship. With the advent of steam early in the nineteenth century, the supply of pure water to ships became a comparatively simple matter, and to-day drinking water on board causes little uneasiness. Great care is, however, taken to eliminate every chance of pollution. All water obtained from shore is chlorinated, and tank-cleaning is carried out by a medically examined party clad in clean overalls, suits and footgear.

The use of steam also to a large extent remedied the food problem by the reduction in the duration of voyages which it made possible. It was on ship ventilation, however, that the greatest benefits were conferred, but, at the same time, encroachment on internal space by the stokeholds and boiler-rooms was made.

Apart from ports and hatches, the wind-sail was for a long time the only aid to ventilation; of limited use in sailing ships, it is still a valuable auxiliary method in steamships, especially in tropical climates. The wind-scoop was not introduced until 1870.

The substitution of fixed cowls for the wind-sail, an idea borrowed from the French, was the next step, but though used early in the nineteenth century, they only came into general use with the building of iron ships; later they were displaced by mushroom top and water-excluding ventilators. The replacement of wood by iron in ship construction was a tremendous advance and brought about many changes, two of which were of great hygienic importance, viz.: (1) The much greater subdivision of internal space. (2) The control of temperature between decks.

Wood is a poor conductor of heat, consequently ships built of this material are not so much influenced by changes of temperature as those built of iron, which is a

good heat conductor. In this respect the wooden ships had an advantage, but, on the other hand, they were often leaky, damp and cold, as, on account of the risk of fire, they were seldom heated. In iron ships, however, owing to heat conduction and convection currents of warm air, the difficulty is to keep them sufficiently cool in warm climates.

This question of heat control has not yet been finally solved, though a great deal has already been done.

The late eighteenth and early nineteenth centuries saw many ingenious mechanical contrivances devised for the purpose of improving ventilation, but owing to the amount of labour involved in their use they were abandoned as impracticable and are of little more than academic interest.

In the early days of steam, attention seems to have been concentrated rather on mechanical attempts to exhaust the foul air from ships than on efforts to supply fresh. About 1850, special shafts, in which lamps or stoves were lighted, were introduced to create an artificial updraught. Steam jets from the boilers were also utilized, by leading them into cowls and ventilating shafts. The most valuable method of exhaust was however, that into the funnel from the ships' furnaces, and this was further utilized when the funnel casing was introduced.

In the meantime, attempts had been made to carry air to various parts of the ship by air-trunks from the cowls, which of course solely depended on the force of the wind. When, therefore, in 1867, steam fans were first used to suck in air, the efficiency of this method was much improved. The introduction of electric fans at the beginning of the present century has done a great deal towards solving the ventilation problem in ships. It is now possible to draw large quantities of fresh air through long systems of trunking and to deliver it in any part of the ship where it is required. Exhaust ventilation is provided for in a similar manner, but owing to the possibility of drawing air from undesirable places, care must be taken that the exhaust capacity does not exceed the supply. The modern tendency is to provide for single compartment ventilation, so that the special requirements of any one compartment can be met without interfering with others.

In naval ventilation there are many factors to contend with which are not experienced to the same extent ashore, and one has to remember that ventilation in ships is largely a compromise. Natural ventilation is of course quite unreliable, except under specially favourable conditions. The subdivision of internal space, the position and small size of some compartments, the limited cubic and superficial space, the absence of scuttles (in some ships), the airtight and heat-conducting material of which a ship is built and the length and bends of ventilating trunks are some of the factors which make ventilation difficult.

Shore standards of cubic and superficial space are quite impracticable, and though minimum requirements are laid down by the Board of Trade for the Mercantile Marine and also by the Colonies and some foreign countries, in the Navy afloat there is no definite floor space per man, but as far as possible 200 c.ft. are allowed.

The deck space only permits of 18 in. between hammock hooks, so that opportunities for spray infection are constantly present.

Epidemiological needs are not satisfied, and considerable ingenuity is therefore required, in the face of a threatened epidemic, to make the best use of available space.

The limitations of cubic space have to a large extent been overcome by the use of electric fans which can supply large volumes of air (heated if necessary), and fans are now provided capable of supplying anything from 500 to 4,000 c.ft. of air per minute.

The great objection to large quantities of air, especially in cold weather, is of course the draught caused. No one will endure a draught however scientifically

produced, and it was found that fans were frequently stopped or air inlets plugged up.

To lessen draught, various devices have been adopted, e.g., the use of deflectors or baffles in the trunking system, directing the air into corners or unoccupied parts of the mess deck—or bringing the inlet to within a foot or so of the deck—to all of which there are objections. The latest method, known as the punkah louvre system, consists of a ball-and-socket arrangement capable of movement in any direction, so that the opening in the ball, through which the air passes, can be directed anywhere. Behind the opening is a rotary shutter, which can be opened or closed at will by means of a small screw.

With a little care each opening can be regulated so that though the general air supply and air movement in a compartment is not interfered with, draughts can be practically abolished.

Heat—the so-called "wild heat"—is another difficulty, particularly in warm climates. Modern requirements have greatly increased sources of heat production in ships and such places as bakeries, galleys, bathrooms, laundries, distilled water tanks, hot food ovens, are now added to the engine and boiler rooms. This problem is being tackled and much has been done to lessen heat conduction from these places and to bring living and enclosed spaces within the comfort zone. The new punkah louvre system and the provision of table fans are of great value in diminishing the ill effects of hot stagnant air. Lagging of bulkheads, steam-pipes and other heat-conducting agencies is helpful, while oil-fired galleys and electric bakeries are also being adopted.

Dampness, principally between decks, is yet another important hygienic matter and one which is closely associated with ventilation. When one remembers that the respiratory and dermal contributions to atmospheric moisture amount to about 45 ounces per man each day and that in addition there may be "sweating" from the ship's side, deck scrubbing, clothes washing, or accidental entry of sea water, it can be realized that the relative humidity on the mess decks is often very high.

The importance of dryness is, however, being recognized, and the provision of bathrooms where clothes can be washed and of special drying rooms is a great advance, but there is still a good deal of unnecessary sluicing down when mess decks are washed. Deck cloths, too, are often laid on the wet deck and, by checking evaporation, tend to retard its drying.

In connection with dust prevention, vacuum cleaners are appliances which may have possibilities.

Food probably more than anything else was the cause of discontent and trouble on board ship in former times, and even to-day a sailor resents much interference with his victuals.

Naval records give us the earliest accounts we have of the feeding of seamen, and from what we read there seems to have been plenty of ground for complaint. As far back as the time of Henry VII, some system of naval victualling existed and the records of the East India Company show that about the beginning of the seventeenth century a fairly extensive diet was at times provided for their ships.

Salt fish, bread and wine were the staple food of sailors until nearly the end of the sixteenth century. About that time salt beef and beer were introduced and, later, this diet was increased by the addition of pork, peas, biscuits, butter and cheese. The beer ration of a gallon a day seems enormous, but it must be remembered that the beer replaced wine and that water was often undrinkable; a five or six weeks' supply was usually taken. About 1690 the beer had become so poor that the Government began brewing it themselves and continued to do so till 1833, when the ration was abolished, but, before its final abolition, brandy, wine and rum had been in alternative use for some time.

At first each man received half a pint of neat spirits per day, but in 1740

Admiral Vernon adopted the practice of adding water before issue, an important hygienic step at this period.

In 1824 the ration was reduced to quarter of a pint and in 1850 to one-eighth pint, which is the quantity still allowed to ratings who are eligible and wish to take it up.

Provided it was all edible, the food issued would supply the necessary calories, but it contained no fresh vegetables, had a high protein content, and must soon have become monotonous. For close on 200 years there was little change in the Navy's food, but towards the end of the eighteenth century improvements were gradually introduced.

Lime juice, as already mentioned, was sanctioned as an Admiralty issue in 1795, while two years later tea was substituted for the afternoon spirit ration.

About 1825, fresh meat, vegetables and sugar were added, while bread, if obtainable, was allowed in lieu of biscuit: chocolate was also issued. About 1850, preserved potatoes were supplied, and nine years later there was an increase in the issues of bread or biscuits and sugar.

In 1867 preserved meat, which had been previously tried in 1814 and in 1850, was again introduced as an alternative to salt beef or pork, when fresh meat was unobtainable. Salt beef was finally abolished in 1907, and nineteen years later salt pork disappeared from the naval dietary.

This 1867 ration remained unaltered for forty years, but in 1907, as the result of investigation by a special committee, a standard ration, plus a messing allowance, was brought in. This allowance, which could be used to supplement the ration as desired, permitted more variety in foodstuffs, and thus eliminated the monotony which had been a feature of previous dietaries.

The principal changes made were reduction in the quantities of bread and meat, increase in the amounts of sugar and vegetables, and the addition of tinned milk and jam or marmalade. This ration, with a few minor alterations, such as a decrease in the amounts of bread and sugar, and an increase in the messing allowance, is the one now issued.

Certain alternatives are provided, if bread, fresh meat or vegetables are not available and small modifications in the diet are allowed at times. Extra issues may be made if special work has to be done, or if climatic conditions demand it, and special provision is made for feeding boys. The present ration is sufficiently varied and elastic to meet most requirements, and, with the messing allowance, will provide the calories and vitamins necessary for health.

Besides improvement in the quality and variety of the ration, the methods of cooking and storing food have changed a great deal. Formerly the position of cook required no special qualifications, but to-day both the cooks and bakers in ships are specially trained men.

Messing conditions are also changing, and the old individual mess is gradually being replaced by general messing. In the old system, the mess appointed one of their number to draw and prepare their rations and take them to the galley for cooking. This system, though it allowed of more individual choice and management as regards food, restricted variety to some extent, and was open to the objection that food was exposed on the mess deck and subjected to unnecessary handling.

The general messing is in charge of an Accountant Officer, who receives special instruction in catering, and is thus in a better position to buy cheaply and in greater variety.

The food is all prepared by trained men, and various mechanical aids—such as dough-kneaders, bacon-slicers, potato-peelers and dish-washers—are utilized. All unnecessary handling is thus eliminated, and, though the messing allowance is lost to the individual, he probably gets better value for his money.

In conclusion I may mention that the replacement of coal by oil has been of

great hygienic advantage in ships. Coaling was strenuous work and often productive of injuries: the crew and ship both became dirty and the subsequent washing down tended to increase dampness on board. Since the introduction of oil, the lot of the stoker has become much easier, and much expenditure of energy and water has been saved. Cases of oil dermatitis crop up now and again, but are few compared with the burnt feet and other injuries met with in coal-fired ships.

Fleet-Surgeon W. E. HOME said that in studying the evolution of naval hygiene, it was surprising to realize how lately it was that hygiene and the prevention of disease had become scientific. At the beginning of the Crimean War, Dr. Dumbreck was sent to make a medical survey of Servia; he could merely report on unhealthy places that they were "wet" or "filthy." Dr. Linton, knowing that malaria came from the air of decay in marshes, recommended encouragement of "the natural respirator—the moustache." Diseases were now individually prevented, thanks to the pathologist, not to the clinician. Malta fever was differentiated from typhoid by Marston in 1860. When he (the speaker) went to Malta in 1886, Malta fever was still thought to be due to chill. In 1887 Bruce found the germ. Ten years later, by the development of the Widal reaction, diagnosis became possible: in 1904 Bruce and the Commission found the cause and stopped the fever. A period of incubation, concealing the date of infection, as in malaria, hampered the study of ætiology and therefore prevention. The deficiency diseases had long incubations; cancer, accordng to Professor Boycott, had a still longer one.

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Section of Laryngology.

President—Dr. DAN MCKENZIE.

[*November* 1, 1929.]

Further Report of Case previously Shown.

Lupus of Nasal Septum.—HAROLD KISCH, F.R.C.S. (shown May 3, 1929)[1].—The anterior half of the septum was involved.

The patient has been treated by diathermy, and there is now no sign of the disease. There is a small perforation. The tonsils were removed subsequently to the diathermy, but showed no evidence of tuberculosis.

The PRESIDENT said that for cases of limited localized lupus, diathermy answered very well, in his experience. He was less sure as to the value of its action when the condition was widespread.

Retro-Orbital Abscess Secondary to Acute Ethmoiditis in a Child.—W. S. THACKER NEVILLE, F.R.C.S.Ed.

PATIENT, a boy, aged 3 years and 9 months.

September 2, 1929, first day of illness, temperature rose to 101°. Family doctor diagnosed intestinal toxæmia and ordered castor oil. Child was obstreperous when castor oil was being administered and banged his eye on the cup.

September 3, 1929.—Left eye proptosed and fixed and both upper and lower lids were œdematous. An ophthalmologist was consulted, who questioned as to possibility of sinusitis, but was told by patient's father (a laryngologist) that nose was normal.

September 4.—The ophthalmologist (Mr. Fison) suspected presence of pus and explored orbit through conjunctiva, but no pus was found.

September 5.—Eyelid of right eye became œdematous. Fundus of left eye showed papillitis and dilated veins and a skiagram showed pansinusitis of left side.

The history was then reviewed and the child's nurse reported that he had complained of face-ache on the day preceding the commencement of the illness, and it was remembered that he had a slight watery discharge from the left nostril and left eye.

Operation (same day).—Patient was anæsthetized and I raised the left middle turbinate. Pus filled the nose, and I then removed the left-middle turbinate and opened the sphenoidal sinus and also opened the maxillary sinus intranasally.

September 6.—Temperature normal. General condition good.

September 8.—A brawny swelling appeared on the inner half of the lower eyelid. The ophthalmologist said that the eye was displaced upwards, and that he could not see the optic nerve; the whole fundus was cloudy. I therefore made an external incision at the inner angle of the orbit and found a large abscess with a dark-coloured perforation of the inner orbital plate. I made a wide opening into the nose and inserted a drainage tube into the orbit. The child quickly recovered, though there still remains an œdema of the lower eyelid.

1 See *Proc. Roy. Soc. Med.*, 1929, xxii (Sect. Laryng.), 89.

The remarkable features of this case were the absence of marked pain and the fact that toxæmia was the first feature noted. The œdema of the opposite eye made one suspect sinusitis. A skiagram was of value ; and that it should be from the case of a child aged 3 years 9 months agrees with the experience of other writers.

It is well known that exophthalmos may be the first sign of an ethmoiditis in children. Congestion of the orbit, due to ethmoiditis, produces exophthalmos, chemosis and œdema. Limitation of movement, dilatation of the veins of the fundus and diminution of vision ought to make one think of an abscess.

Retro-orbital Abscess Secondary to Acute Ethmoiditis. (Mr. Thacker Neville's Case.

Discussion.—Mr. W. STIRK ADAMS asked for further details about the fixation of the eye, because in a recent case fixation of the eye plus central proptosis was diagnosed by an ophthalmologist as an intra-orbital condition. Was the proptosis in this case absolutely central? It was important to know whether central proptosis could occur as the result of subperiosteal abscess on the ethmoidal side.

Dr. J. ALDINGTON GIBB said that sometimes proptosis had no connection with œdema, but it was due to paresis of ocular muscles. Sometimes the eye was pushed on one side owing to strabismus. Often proptosis was due to Tenon's capsule becoming inflamed. In a case of proptosis without œdema of the eyelids, he did not think one was justified in concluding that the condition was either orbital cellulitis or suppuration.

With regard to operative procedure, each case must be approached by selection. In the severe cases external approach was usually to be preferred. The milder cases might be attacked by the intranasal route.

Sir StClair Thomson said that Professor von Eicken appeared to treat such cases surgically by passing through the os planum of the ethmoid into the orbit. That seemed to him (the speaker) a perilous way of approach, and he would think if there was a retro-ocular abscess it would be safer to open it from the outside.

Mr. E. Craig Dunlop said he had had a series of these cases, and in many of them the os planum was already perforated, and there was no need to make a hole. They could be successfully drained by the intra-nasal route. Displacement was not reliable from the diagnostic point of view. Sometimes there was lateral displacement of the eyeball, sometimes forward only. The results of drainage by the intranasal route were good. In one of his cases he had removed some orbital fat, which was protruding through a hole in the os planum, and of course through the periosteum as well.

Dr. J. S. Fraser said that all cases of acute proptosis were not due to sinusitis. Recently in hospital he had seen a case of proptosis sent over from the Ophthalmological Department with the eye proptosed. The swelling appeared to be behind and to the outer side of the eyeball. The eye specialist thought it was an orbital complication of nasal sinus trouble, but there was nothing wrong in the nose. Transillumination of the frontal and maxillary sinuses was favourable, and radiography in every direction showed healthy sinuses. Dr. Logan Turner saw the case with him, and agreed that nothing wrong was to be seen in the nose by rhinoscopic or radiographic examination. He (Dr. Fraser) only learned the day before he left that the Wassermann reaction was positive; it was probable that there was some gummatous infiltration at the back of the orbit.

Mr. W. S. Thacker Neville (in reply) said that the abscess was between the periosteum and the os planum and the fixation of the eye was probably due to œdema. The temperature was raised on the first day and on the next day the eye was proptosed, but there was no strabismus. The ophthalmologist had no difficulty in seeing the optic nerve each day, and no displacement of the eye was reported until the seventh day, when a brawny swelling of the lower eyelid appeared and the eye was displaced upwards so that the optic nerve could not be seen. It was then decided to perform an external operation.

Postscript (December, 1929).—The œdema has disappeared.

Carcinoma of Right Bronchus.—W. S. Thacker Neville, F.R.C.S.Ed. Patient, male, aged 64, a wagon builder and upholsterer, came on July 26, 1929, complaining of a cough, which continued almost incessantly. He had been expectorating liver-like substance with streaks of blood for three or four months, and one hour after eating he vomited. He used to weigh 11½ st., and then weighed 10 st. 10 lb.

On examination the larynx was normal. A rubber tube passed into the stomach without any obstruction and so a skiagram was taken. This showed atelectasis of the right upper lobe. On July 29 I passed a bronchoscope half an hour after the administration of morphine hydrochloride gr. ½, with atropine gr. $\frac{1}{75}$ and the cocainization of the larynx. At the entrance of the right bronchus I found a tumour, which moved with respiration. After withdrawing the bronchoscope the patient coughed up a large piece. On section this was reported to be an oat-celled carcinoma, and on July 30 I injected both lungs with lipiodol, and found that the lipiodol did not pass into the upper right lobe. I then procured a 50-mgm. tube of radium from Dr. Gamlen, of Newcastle, and inserted it into the right bronchus. This resulted in atelectasis of the whole right lung as seen by X-rays. The patient was relieved to a great extent of his cough, but rapidly lost flesh and died three weeks later.

The patient's symptoms were cough, dyspnœa, expectoration, hæmoptysis, loss of weight, and fever, and so one sees how easily one could diagnose such a case as

tuberculosis of the lung. When I first saw him, I thought he had a mediastinal tumour or an aneurysm, and I did not suspect a bronchial tumour until I saw it with a bronchoscope.

Atelectasis seen in a skiagram suggests the presence of a tumour; it is the commonest sign of—and in many cases the only clue to—malignancy. In this case there were no signs of suppuration, such as have been reported by other writers, and so I did not think of abscess or bronchiectasis of the lung.

The malignancy of this tumour is classified by Dr. F. B. Smith, of Harrogate, as No. 3 Broder, that is to say, it was highly malignant. This agrees with the findings of most writers, although some have reported patients alive four and eleven years after the removal of malignant tumours.

The treatment in this case was radium; the usual intensive X-ray treatment was not at my disposal.

Discussion.—Dr. BROWN KELLY said there were several features in this case which suggested a diagnosis other than that given. Thus, it was stated that the tumour at the entrance to the right bronchus was expelled on introducing the endoscope and that on inspecting the region afterwards no growth was seen. The microscopical characters of the piece expelled were unusual. Was this really a tumour? He asked whether the possible presence of an abscess in the liver had been considered. This could make its way into the right lung, whence its contents would be coughed up. The discharge would resemble the liver-like substance expectorated by the patient. In one of the skiagrams the diaphragm was higher on the right side.

Mr. V. E. NEGUS said that a point brought home by this case was the necessity of examination with the bronchoscope in all cases in which carcinoma of the lung was suspected. He had had two cases in which this did definite good. One was that of an elderly man, whom Sir StClair Thomson allowed him to examine. There was an obstruction of the right bronchus by masses of new growth; bronchoscopy made the diagnosis definite, and the clearing away of the masses afforded considerable comfort, though the patient died afterwards. The second case was one which Miss Muriel Ward had allowed him (the speaker) to see. There was a new growth in the right bronchus which had caused collapse of the right lung. There was no air entry owing to the mechanical obstruction caused by the growth. He cleared away a good deal of pus, as it was better to carry out removal after this procedure. He then removed the growth as a single tumour, pedunculated. An excoriated area was left on the bronchus and this he painted with silver nitrate. The growth was an adeno-carcinoma, and the man was apparently well for the time being; the skiagram showed the lung to be clear, there were no symptoms and the man had gone back to work. Probably many cases of carcinoma in the lung originated in the bronchus, whence it was possible that growths might be easily removed. Even if this patient should have a recurrence he was rid of the trouble for the time.

The PRESIDENT said that carcinoma of the lung seemed to be increasing, and if that were so, one wondered to what the increase was due. A collection of cases, with full details, would be of considerable interest. If cases could be seen and diagnosed early, radium or other means might be applied with some prospect of benefit.

Mr. W. S. THACKER NEVILLE (in reply) said he did not think the condition was liver abscess, as both Mr. Barnard and Dr. Smith reported carcinoma. Carcinomata of the bronchi were not very well classified. Mr. Negus's case appeared to have been like this one; it was strange that the right lower lobe was not obstructed.

Chevalier Jackson stated that formerly 1% of primary carcinomata were in this situation but that, lately, primary carcinoma of the bronchus had increased 15% to 20%, giving as the probable reason the petrol and the tar used on the roads. The only way to diagnose the cause of a chronic cough was to pass a bronchoscope, and this procedure could be rendered more acceptable by using local anæsthesia.

Three Cases of Non-Opaque Foreign Body in the Right Bronchus.—V. E. NEGUS, M.S.—(I) Male, aged 54. Cough with much sputum for about sixteen months.

Rabbit vertebra removed at second bronchoscopy, with local anæsthesia.

(II) Girl, aged 6.—Piece of Brazil nut in right bronchus causing ball valve obstruction. Removed, with local anæsthesia, twenty-four hours later. Almost immediate cure.

(III) Nut, from milk chocolate, in right main bronchus, for twenty-one days, causing ball-valve obstruction. Right bronchus cleared by suction through bronchoscope, under general anæsthesia. Signs cleared up rapidly. Child became perfectly well.

Discussion.—Sir JAMES DUNDAS-GRANT said he had had a case in which there was a rabbit's vertebra in the left bronchus. The foreign body was sufficiently opaque for the skiagram to show it fairly well, and he extracted it.

Mr. M. VLASTO said that during the ten years that he had been on the staff of the Queen's Hospital for Children, he only remembered one case which he had been asked to investigate for a possible non-opaque foreign body. The patient was a young child with extensive signs in the left chest, chiefly affecting the upper lobe. The child's general condition was precarious. His temperature had been swinging for weeks, and he had a very high leucocytosis. There had been no history—at the time—of the aspiration of a foreign body. It was only later that the parents remembered that the child had choked when eating peanuts, some considerable time before he was taken ill.

As soon as the bronchoscope had penetrated the left main bronchus, there was a fit of coughing with the expectoration of a quantity of pus and débris of organic matter. Dr. Owen, who was at the moment auscultating the chest, advised him (the speaker) to cease instrumentation because he could now hear bubbling breath sounds coming through over the area which had hitherto been silent. The episode was rather dramatic, because the patient after being almost moribund made a complete recovery.

The PRESIDENT said the moral of these cases was that the idea of a foreign body should occur to the mind of the physician—possibly also to the mind of the laryngologist—more frequently than it did.

Dr. T. B. JOBSON asked whether an asthmatoid wheeze had been present in the cases in which there was obstruction. He (the speaker) had reported a case, during the previous year, in which that clinical symptom had been evident. It was that of a child with a foreign body —a flake of bone—in the left bronchus, and the "wheeze" had put him—Dr. Jobson—on the right track.

Mr. A. LOWNDES YATES said that he had had a case which showed how small a foreign body could cause obstruction to a bronchus. The son of a doctor was running along a hedge with a stick in his mouth, when he bit off a shred of wood from it and inhaled it. The result was a lung abscess, and only with difficulty was the foreign body removed with forceps, after the pus had been aspirated from the abscess, the walls of which were formed by the bronchial tubes. These were enormously dilated, although the foreign body had been in position for only three days. This was the more remarkable in view of the very small size of the foreign body, which was only of the thickness of paper, $\frac{1}{4}$ in. long and $\frac{1}{4}$ in. wide. He took it that the bronchial tubes beneath even this very small obstruction filled up with fluid very rapidly in this case—and probably in other cases—and that permanent damage might be caused with great celerity.

Mr. F. C. W. CAPPS spoke of a child, aged 6 months, previously healthy, who had been playing with a pea-pod. As he presently had a choking fit, the mother considered that a pea had been inhaled. When brought to hospital the child was distressed and cyanosed. Screening with X-rays gave no sign. The physician said there was no air entry on the left side. He (the speaker) passed the finest tube which he had down the trachea, and directly it reached the bifurcation, though no foreign body was seen, the child breathed well. The entrance to the left bronchus seemed rather red and pouting. As the instrument was withdrawn the child vomited a pea, which had probably been lying in the œsophagus, and had obstructed the left bronchus from behind. The child died 24 hours later, and was found to be riddled with miliary tubercle.

Mr. V. E. NEGUS (in reply) said it was sometimes difficult to get physicians to allow cases to be examined in this way. In many cases of lung disease and lung suppuration he regarded

the bronchoscope as of equal importance with the stethoscope. He would consider the first foreign body—as compared with Sir James Dundas-Grant's case—relatively non-opaque; against the collapsed lung tissue it did not show.

In regard to Mr. Vlasto's case, where there was a vegetable foreign body one must not concentrate just on the piece of foreign body, but must think also of the local swelling of the mucous membrane. In many cases there was a violent tracheo-bronchitis, which necessitated tracheotomy. In long-standing cases it was the granulations which blocked the bronchus, not the foreign body. The small piece of nut originated a great deal of swelling, and not until that piece had been removed did the swelling disappear and the child get well.

In answer to Dr. Jobson, the right bronchus was concerned and there was no air entry, and in such cases no breath sounds were heard. To cause asthmatoid sounds there must be some passage of air. In lung abscess or bronchiectasis there might be obstruction owing to formation of granulation tissue, and much improvement was effected by getting rid of the granulations. In answer to Dr. Capps, the foreign body in his case had been only a small one; a large one might press on the trachea. The œsophagus should be examined too; in some cases it was difficult to differentiate as to whether the foreign body had been swallowed or inhaled.

Disappearance of Epitheliomatous Outgrowth of Pharynx after Application of Diathermy.—DAN MCKENZIE, M.D.—Male, aged 52, first seen at the Central London Throat and Ear Hospital, May 27, 1929, complaining of sore throat and difficulty in swallowing, of twelve months' duration.

A large, smooth, rounded tumour was attached to the posterior pharyngeal wall towards the left of the middle line, and lying over the larynx in such a position as to prevent a view of the interior.

On June 4 a piece was removed for examination, and the tumour was traversed in several directions with a live diathermy electrode.

The pathological report (G. Scott Williamson) showed the growth to be an "epithelioma of basal-celled type."

As the application of diathermy was followed by a shrinking of the growth to a mere nodule on the pharyngeal wall, this nodule was itself submitted to diathermy on June 25. By this time the vocal cords had become visible, and the left cord was seen to be fixed. On July 8 this cord was moving and the nodule had disappeared.

Up to the time of writing (September 24), there has been no sign of recurrence, but the future is dubious, of course.

The sudden and apparently complete disappearance of the large neoplasm resembles the surprising change that sometimes follows the use of radium in pharyngeal sarcomata, but this is the first time I have seen that event following diathermy.

Discussion.—The PRESIDENT said that with regard to the diagnosis of basal-celled carcinoma of the pharynx, pathologists were making that diagnosis more frequently than formerly, and he asked what was the relative malignancy of these cases; was it about the same as that of rodent ulcer? Did it affect the glands? And what was the outlook for the patient?

Sir JAMES DUNDAS-GRANT: They answer to irradiation better than prickle-celled epitheliomata.

Diathermy Removal of Soft Palate for Epithelioma.—DAN MCKENZIE, M.D.—Male, aged 58. Operations in February, 1929: (1) Removal of cervical glands and ligature of external carotid. A chain of glands under the sterno-mastoid necessitated removal of part of the muscle. Subsequent serial examination by G. Scott Williamson, however, revealed no sign of new growth in them. (2) Removal of soft palate ten days later. The growth was extensive, involving the left side of the palate and spreading back on to the lateral wall of the pharynx.

The consequent gap is closed by an obturator attached to a denture.

Extirpation of Tonsils by Diathermy-Coagulation.—DAN MCKENZIE, M.D. Tonsils very large, removed by weekly applications extending over six months. Patient, a subject of exophthalmic goitre, shows considerable improvement in symptoms.

Dr. J. S. FRASER asked what was the object in using diathermy in the second case; there remained some tonsils on both sides. The case seemed a suitable one for dissection. He did not think anything was gained by taking a long time over the operation.

Mr. W. S. THACKER NEVILLE asked whether there was any œdema of fauces or palate. He did not think age constituted a reason for not dissecting out the tonsil. He himself had dissected out—under local anæsthesia—the tonsil of a woman aged 72, on account of rheumatism.

Mr. T. RITCHIE RODGER said he thought the injected appearance of the anterior pillars showed the tonsils to be still septic.

Mr. W. STUART-LOW said that he had used the galvano-cautery in preference to diathermy; it was safer and less alarming to the patient.

Mr. E. CRAIG DUNLOP asked how the decision was reached that the deeper parts of the septic tonsil had been removed. The serious sepsis could not be exterminated by removing the protuberant part of the tonsil. The sepsis was chiefly in the deeper part of the crypt, and there appeared to be some tonsillar tissue remaining, he would therefore like to know whether the exhibitor proposed to go further.

Dr. T. B. JOBSON said that, so far, he was favourably impressed with this method. The cases for which he particularly chose it were those with infective arthritis, and those with a flat septic tonsil. After tonsil enucleation there was often a flare-up of the rheumatic condition. He did not understand why Dr. McKenzie was not a little more strenuous in his application, because most of the greater portion of the tonsil could be removed by two or three applications, most of it at the first sitting. In three or four weeks the removal could be completed. He had never had to wait six months for that result. There was little swelling of the fauces, and it was a satisfactory method for a particular type of case.

The PRESIDENT, in reply, said that the use of diathermy in the second case was by way of experiment. The applications were all made under local anæsthesia. The difference between the galvano-cautery and diathermy was very great. After the use of the former there was scarring and a closure of the crypts, but after diathermy there was no scarring of the crypts. He preferred to make small advances so as to minimize risks of hæmorrhage. For patients who were unsuitable for operation he was sure diathermy coagulation would become the routine treatment.

Epithelioma of the Vocal Cord (with Illustrations showing the Condition of the Larynx during the Application of Radium).—HAROLD KISCH, F.R.C.S.—Patient, male, aged 62. History of six months' hoarseness following cough and cold.

On Examination.—Warty growth of right vocal cord, with hyperæmia and a very slight hesitation in movement. Tubercle and syphilis were excluded in the usual manner, but no portion of the growth was removed for examination.

On July 9, 1929, five needles of radium, each containing 1 mgm., were inserted on the outer side of the larynx, through a window made in the thyroid cartilage. The skin incision was completely closed; no tracheotomy was performed. The needles were removed after five days, the total dose of radium being 600 mgm. hours.

The patient was very tolerant of examination, so that accurate drawings could be made during the whole period of treatment. (See figures p. 8.)

On September 31, 1929, the right cord showed some hyperæmia and slight œdema, but there was no sign of the growth.

I am indebted to Miss Hulke, House-Surgeon at the Royal Ear Hospital, for the drawings for these illustrations.

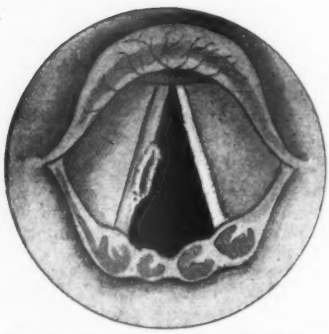


FIG. 1.—Before insertion of radium Cord partially fixed. (9.7.29.)

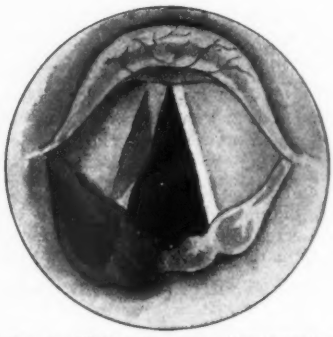


FIG. 2.—Thirty-six hours after insertion of radium. Cord immobile. (11.7.29).

FIG. 3.—After 3 days. Cord immobile. (12.7.29.)

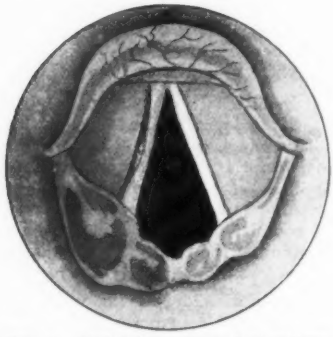


FIG. 4.—Just before removal of radium. Cord immobile. (14.7.29.)

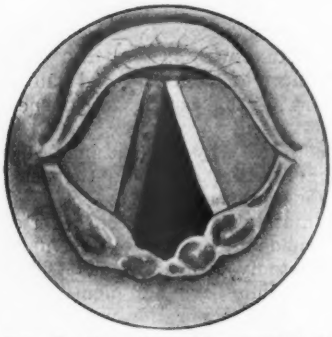


FIG. 5.—Four days after removal of radium. Cord quite mobile. (18.7.29.)

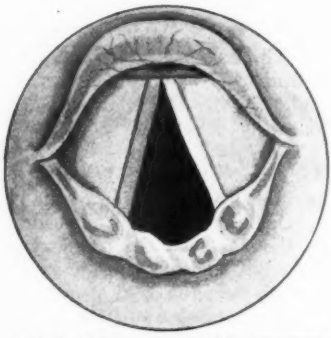


FIG. 6.—Three months after treatment.

Epithelioma of the Vocal Cord: showing the condition of the cord during the application of radium. (Mr. Harold Kisch's case.)

Discussion.—Dr. J. S. FRASER said these cases ought to be followed up and reported on regularly. Three had been reported by him at a recent meeting in Scotland, and he had since then treated three others. If all cases were followed up as Sir StClair Thomson's were, one could then compare the results ot treatment by laryngofissure with those obtained by radium therapy.

Sir STCLAIR THOMSON said that he had had some cases, but he did not himself apply the radium. He would say there were some "misses" as well as some "bulls' eyes." Some patients had declined laryngofissure or laryngectomy, though the cases had seemed favourable for operation. After radium treatment the condition had cleared up as if by magic.

One case was that of a medical man who had declined treatment by operation. He returned to his practice with a full voice, but within two years he died. In one of these cases treated by radium the history went back very far. It was necessary to wait until at least three years had elapsed since the treatment. Restoration of voice was a small matter in comparison with saving life. He had shown before the Society four medical men who were still alive and well at four, five, seven and eight years after operation, with voices sufficiently good to enable them to carry on their practice.

It was instructive to see the drawings which had been exhibited, as they showed that the action of radium was not limited to the cords. It would be necessary to find out afterwards what was its action on the larynx. In Germany and America there was literature showing the disastrous late effects of irradiation on the cartilages of the larynx. Unless laryngologists agreed to publish all their cases there would not be much progress.

Sir JAMES DUNDAS-GRANT said there was a good case for thyrotomy when the disease was limited. He had just heard from a patient on whom he had operated sixteen years before for epithelioma of the vocal cord and who was still quite well and talkative.

Dr. ANDREW WYLIE said it was satisfactory to hear such good results following radium treatment for epithelioma of the larynx, but what proof had been furnished that the condition in this case was epithelioma? It seemed to him to be a small warty growth on one cord. He believed in always having a piece removed for examination. He was aware that Sir StClair Thomson did not always agree that this should be done, but as he (the speaker) had once made a serious mistake, he considered it absolutely necessary, and if a piece had been removed in Mr. Kisch's case and reported as malignant, the diagnosis would be complete.

Mr. HAROLD KISCH (in reply) said he must remind Members that he was not sure it was epithelioma, but it was so clinically. If radium were never tried in such cases no conclusion could be arrived at as to whether it was of any use. Not much damage was done by it even if it failed, because removal by surgery could still be carried out. The object of applying radium was not only to affect the part itself, but to cut off lymphatic infection. The fact of the whole larynx being affected was a strong point in favour of using radium. A screen of one-fifth mm. of platinum formed the screen. He had used it now in three or four cases in five years, and he would try to collect the particulars. The case was shown to demonstrate, by pictures drawn from life, the naked-eye appearances of what occurred in the larynx during the application of radium.

Foreign Body (Halfpenny) in Œsophagus removed by Jackson's Œsophageal Speculum.—W. S. THACKER NEVILLE, F.R.C.S.Ed.

I believe this to be the most suitable instrument for removing a foreign body hidden behind the crico-pharyngeus muscle.

The patient was a child, aged 5 years, who had swallowed a halfpenny four days before admission to hospital. He was able to swallow food in spite of the presence of the halfpenny, but he was brought to hospital on account of a commencing dysphagia.

A skiagram was taken and the halfpenny was lying placed frontally at the upper end of the œsophagus. An œsophageal speculum was passed and the usual prominent muscle known as the crico-pharyngeus at the upper end of the œsophagus had to be pushed aside in order to expose the halfpenny which was hidden by the muscle and

tightly wedged in place. A foreign body in such a place can easily be overridden by an ordinary œsophagoscope, whilst this instrument allows one to use a powerful leverage and so expose the halfpenny.

The headrest was that used in Haslinger's Hajek clinic, which allows one to place the child's head in Jackson's position, without the aid of an assistant.

Laryngeal Case for Diagnosis.—G. BOTROS, M.B., Cairo (introduced by Mr. T. B. LAYTON).—S. S., male, aged 49, referred to Guy's Hospital, September 16, 1929, on account of hoarseness and discomfort in swallowing for the last eight weeks.

On Examination.—Voice strong but slightly husky; slight discomfort referred to left side of throat, mostly marked in the morning, passes off later in the day. No pain, cough or dyspnœa.

Larynx.—Mucous membrane of right arytenoid and laryngeal surface of interarytenoid region is slightly congested and studded with several small papilliform points. Left ventricular band is swollen and the overlying mucous membrane is smooth and slightly redder than normal. On the posterior extremity of the band is an ulcer 6 mm. in diameter, with irregular margin and shallow floor, covered with a greyish membrane. No inflammatory areola. Cords move well. No laryngeal tenderness. No swelling of glands. Wassermann reaction negative. Examination of chest for tubercle, negative.

Patient is a light smoker and drinker.

Family History.—Nothing important.

Discussion.—Mr. HAROLD BARWELL suggested that this was a septic laryngeal condition, secondary to a septic condition in the nose.

Dr. J. S. FRASER said he considered that the diagnosis lay between malignant disease and tuberculosis. He inclined towards the former. It was, however, difficult to decide without removing a piece for biopsy.

Dr. BROWN KELLY said he thought this was a growth of uncertain nature involving the anterior two-thirds of the cord.

Sir GEORGE BADGEROW: I think it is a growth that has become septic.

Dr. JOBSON HORNE said he was entirely opposed to the opinion expressed that the condition in the nose was reponsible for the disease in the larynx.

Sir JAMES DUNDAS-GRANT: A portion should be removed for microscopical examination. I think it is probably epitheliomatous.

Aneurysm, (?) Ascending Pharyngeal.—E. BROUGHTON BARNES, F.R.C.S.Ed.—Female, aged 59. A dilated tortuous vessel can be seen pulsating immediately beneath the mucosa of the lateral pharyngeal wall, behind the left posterior pillar of the fauces. The diameter of the vessel appears to be almost equal to that of an ordinary lead pencil.

Discussion.—The PRESIDENT questioned whether this was aneurysm, and also whether the vessel was the ascending pharyngeal artery. Many years ago he had seen an aneurysm of the internal carotid, but pulsation was greater than in this case. A surgeon opened the neck, and was sorry he did so, as the patient died.

Dr. BROWN KELLY remarked that in the *Journal of Laryngology* 1925, p. 15, reference was made to 85 cases of this kind. The appearance in the pharynx was due to tortuosity of the internal carotid. Every now and then such cases were described as aneurysm. The vessel was not the ascending pharyngeal, which was small and on the posterior wall of the pharynx. He had operated on several of these cases, in which there was a bulging of the artery into the pharynx. The tonsil during removal should be drawn forwards and inwards and thus away from the artery.

Mr. M. VLASTO said that about eight years ago, when he was carrying out a simple removal of tonsils by the reversed guillotine method, there was a sudden gush of blood, and the patient died in a few minutes. The post-mortem revealed a very superficial and tortuous carotid artery. The guillotine blade had undoubtedly been too sharp, and he had removed more of the posterior pillar than was justified.

The PRESIDENT said it was well to know of these accidents, as the knowledge prepared the mind for them and one could then know what to do. The Section was grateful to Mr. Vlasto for having reported such an accident.

Late Result of too high a Tracheotomy.—E. BROUGHTON BARNES, F.R.C.S.Ed.—Girl, aged 17. Emergency tracheotomy when aged 5. Tube appears to have been inserted through the thyroid cartilage and the crico-thyroid membrane. There is a considerable scarring in the glottis, the size of which is much reduced. The right cord is almost fixed, but she has a very good voice.

Sir JAMES DUNDAS-GRANT said that in view of the circumstances attending laryngeal diphtheria, we should be lenient in our judgment when criticizing a practitioner for having made a tracheotomy too high. He had had referred to him a young child in whom this occurred, and he was able to get him well by using intubation tubes larger than were suitable for a child of his age. By means of a nasal tube it was possible to feed the child, who recovered and grew up to be a strong man. In such cases as the present the treatment was to make another tracheotomy opening lower down, and allow the larynx to have complete rest, after which the question of laryngostomy came in, but he thought this patient was doing well enough and would not need further interference.

Hoarseness: Thickening over Right Arytenoid. Case for Diagnosis.—A. L. MACLEOD, M.B.—P. C. B., male, aged 27. Seen in January, 1928, on account of hoarseness of twelve months' duration. Slight cough and expectoration.

On Examination (January 4, 1928).—Thickening seen over right arytenoid. Slight ulceration in front of arytenoid, not on cord. Movements of cord restricted. Sputum, negative. Wassermann reaction, negative. Family history of tuberculosis. Patient has been in hospital at Ventnor for six months, having silence treatment; whispering has been allowed. Discharged three weeks ago—no active mischief.

Present Condition.—Still hoarse. Thickening still seen over right arytenoid, involving aryepiglottic fold and ventricular band; cord still partially fixed.

Discussion.—Dr. JOBSON HORNE said he regarded the lesions in the larynx as due to tuberculosis. He advised treatment by absolute silence in a sanatorium where it could be more easily carried out than in the patient's home.

Sir JAMES DUNDAS-GRANT said examination of the sputum frequently gave negative results until an enforced cough, which could be brought about by sniffing oil of mustard, was produced. If that failed, the administration of iodide of potassium for a few days often brought about a positive sputum.

Laryngo-Scleroma (?). Case for Diagnosis.—R. J. CANN (for Mr. W. M. MOLLISON).

Male, aged 46. Complained of hoarseness three years ago. Attended out-patients' department in 1927. Has had dyspnœa for the past twelve months; this became acute in May, 1929. Tracheotomy performed in St. Giles' Hospital, May 29, 1929.

Wassermann reaction negative.

Sputum examined several times; no tubercle bacilli found.

Sir JAMES DUNDAS-GRANT said there was a growth below the right cord, and he suggested the removal of a portion of it with Mackenzie's forceps, to make a diagnosis and possibly, at the same time, to relieve the breathing.

Postscript.—Microscopical examination of a section showed the growth to be epithelioma. (W. M. M.).

Section of Otology.

President—Mr. W. M. MOLLISON, M.Ch.

[*November* 1, 1929.]

SUBJECT FOR DISCUSSION: TRIUMPHS AND FAILURES OF THE MASTOID OPERATION.

PRESIDENT'S ADDRESS.

A Brief Survey of the History of the Mastoid Operation.

By W. M. MOLLISON, M.Ch.

IN order to introduce the subject of our discussion I have chosen for this address the history of the mastoid operation, or more accurately, a sketch of that history.

Most of us have read—and all of us ought to have read—Sir Charles Ballance's account of the history of the mastoid operation in his "Essays on the Surgery of the Temporal Bone": this is so full and eloquent that it is scarcely decent for me to choose the subject: however, I have shamelessly drawn on his account for the early part of this sketch.

The first reference to the operation is in 1649: Johannes Riolanus wrote:—

"Joining the tympanum towards its upper part there is seen a small and very narrow opening, which, gradually enlarging, passes into a cavity of considerable size, presenting everywhere little depressions, like the cells of a honeycomb filled with air . . . it happens that when this air, which should always be still, is stirred up in the ear by the impact of a fresh-air current, that the ears buzz continually. But how can we give exit to this air which is such a source of trouble? There is certainly no other way of doing this except trephining the mastoid process."

And, again:—

"therefore when this canaliculus (Eustachian tube) has become useless why should not the cellular end which communicates with the ear be made to serve the same purpose by perforating it with a sharp instrument."

Thus we see that the earliest operations on the mastoid were suggested to relieve tinnitus or to make a new Eustachian tube; only later was the operation done to give an outlet for pus.

Though 1649 is given as the date of the earliest operation, it may have been done sooner, and I am fortunate in being able to show you a rare drawing by Van Leydon called "The Surgeon." I am indebted to my friend, Dr. Shackle, for this. It is dated 1524, and represents a surgeon making an incision behind the ear of a patient, seated on the floor in front of him.

A second picture (shown), also perhaps of a mastoid operation. The date of this is about 1650; perhaps we may imagine this was inspired by Riolanus. It is by Jan Havickz Steen.

Apparently, Petit performed the first operation on the mastoid for evacuation of pus—this was probably about 1740. He was an advocate of early operation; he wrote:—

"these abscesses . . . may persist a long time before they cause death; but from the very first days of their formation, they ought to be opened."

He used a trephine, illustrated in Ballance's book.

In 1776 Jasser operated on the mastoid in order to evacuate pus and to cure deafness. His method of operating was to bore a hole in the bone. He then fitted the nozzle of a syringe into this hole and forced in the lotion to drive out the pus. This procedure was repeated on twelve succeeding days. He had some successes.

After this, the operation fell into disrepute owing to the death of Berger, the Danish Court physician, on whom it had been performed on account of tinnitus and deafness. Nevertheless, it must have been carried out now and then, since, in 1821, Itard wrote to condemn it.

In 1838 Dezeimeris wrote in its defence, reviewing several of the previously known cases including that of Berger, and pointing out that the adverse criticism was unfair and that the operation was to be encouraged. It was, however, many years before his advocacy was followed, not, indeed, till 1868 or 1870.

It is of interest to notice that it took centuries to evolve the idea of opening the mastoid process for the release of pus: up to 1870 a few bold spirits only performed the operation of boring the bone to give exit to pus or to rid the patient of tinnitus. Naturally, the absence of anæsthetic and the lack of all knowledge of cleanliness retarded surgical progress; by contrast, it is striking to observe the advanced state of knowledge of anatomy and, to some extent, of pathology.

Here are a few examples: In 1683 Duvernay published a very complete work on the "Organ of the Ear, containing its Uses and Diseases of all its Parts": he described the condition of suppuration in the labyrinth.

In the seventeenth and eighteenth centuries Valsalva wrote his anatomical work; Morgagni, his pupil, pointed out that abscess of the brain was often secondary to disease of the ear, thereby correcting the contrary view held before.

Later, Gull, 1855, gave an excellent description of the pathology of lung infection secondary to lateral sinus thrombosis and showed that this was secondary to disease in the mastoid cells.

This long period of "groping for the operation" as we may call it, was brought to an end by the standard work of von Tröltsch and Schwartze; the von Tröltsch's work, published in 1861, was overshadowed by that of Schwartze to whom is given —almost universally—the credit of the mastoid operation, though it would appear that this country was not far behind, if indeed, at all, in this matter.

His work was published in 1873. There were three great English-writing aurists: Wilde (Dublin), Toynbee, and Hinton, and we may allow ourselves to believe that they too influenced the progress of mastoid surgery. Wilde in his "Aural Surgery," published in 1853, advocated his incision through the soft tissues and periosteum. This book was translated into German and perhaps had a direct influence in leading von Tröltsch and Schwartze to their operation.

In passing, it is delightful to think of the interesting and leisurely existence of the early English writers on the ear; thus Wilde, author of "Practical Observations on Aural Surgery," wrote "A Voyage to Madeira, Teneriffe and along the Shores of the Mediterranean, with Observations on the Present State of Egypt, &c." and "Austria, its Literary, Scientific and Medical Institutions."

Hinton, too, wrote many philosophical works, among others, "The Mystery of Pain."

Wilde's account of his incision is as follows:—

"A perpendicular incision, about 2 in. long, nearly parallel with the posterior border of the auricle, is made by inserting a sharp-pointed scalpel down to the bone at the insertion of the mastoid muscle and carrying it upwards and a little backwards—a piece of lint was inserted, when bleeding stopped, linseed poultices applied—the wound suppurated kindly."—Patient recovered.

In his book published in 1868, Toynbee showed that he realized the importance of opening the bone; he wrote:—

"Perforation of the mastoid process also suggests itself and this operation may doubtless be performed in those cases where the matter is pent up in the cavity of the ear and is causing such urgent and serious symptoms as are likely, if not relieved, to terminate in death. I have never performed this operation but I should not scruple to do so in a case where the life of a patient were threatened . . . the best plan of operating would be to use a trephine over the middle and posterior part of the process and to remove a portion of bone ¾ of an inch in diameter."

Hinton, in his supplement to Toynbee's book, urges Wilde's incision. He also mentions the importance of incising the swelling of the posterior deep meatal wall; but in his book published in 1874, he wrote of inflammation over the mastoid:—

"Free incision, carried completely through the periosteum to the bone should be made in the swollen part . . . but if the division of the periosteum over the mastoid process leaves the symptoms unchecked, the perforation of the mastoid cells should not be delayed more than a day or two. It is easily carried out. Often when pus is contained within the cells the bone is softened and the knife will penetrate it or a strong probe will break it down. When this is not the case a gouge or drill may be used: I employ a drill with a movable guard . . ."

He used the ether spray to deaden the pain.

In America too the operation was performed. In his book, "Diagnosis and Treatment of Ear Diseases," published in 1880, A. H. Buck, of New York, advised operation, and quotes cases of recovery after it. Thus he wrote:—

"The mastoid was opened by means of a drill applied half an inch behind the meatus, a little below the level of the upper wall and directed inwards, decidedly forwards and a little upward. At a depth of ⅜ of an inch the first cell was encountered but no pus found. Two days later free discharge of pus took place."

This he did in 1873.

We thus see steady progress from Wilde in 1853, to Hinton, 1873.

This phase continued little changed for some few years and then the operation was enlarged to include other cases as well as the acute, and Stacke and Jansen then evolved the more drastic operation of complete removal of all the contents of the middle ear, in cases of chronic suppuration: this operation we know as the radical operation and its date of inception was about 1889.

Even before this Küster had suggested the complete operation, but his work was not recognized.

In this country, at any rate, the radical operation was known as Stacke's operation—as a fact it ought to be called Zaufal's operation, Stacke's technique being devised originally for cases in which the lateral sinus occupied a very forward position.

In the light of our present knowledge it is clear that at first many cases were subjected to the radical operation quite unjustifiably and the total amount of hearing thus lost is incalculable. However, this orgy of destruction did not last long, probably not more than seven years; indeed, one of the most satisfactory features of that period—1889-1897—was that those men who first advocated the radical operation were themselves the first to try and save the ossicles. Panse, Scheibe and Jansen were examples. Stacke himself advised conservative operations in 1897, only eight years after he had devised the radical operation. This then initiated the phase of conservatism.

We might express the progress of mastoid surgery in a table of "phases."

Naturally there are no sharp-cut divisions between these phases but there is a gradual improvement in all directions, with many modifications in methods of operating and dressing.

Progress has been intimately bound up with earlier diagnosis and with fuller knowledge of the anatomy through the outstanding work of Cheatle, never properly appreciated outside this country; X-rays have been of some help; how much help we are to discuss at a future meeting. Once the conservative habit had established itself, modifications were suggested.

The survey of the most recent phase begins with Stacke's paper in 1911, on "Conservative Radical Operations"; he pointed out that he had advised the preservation of the ossicles in certain conditions as early as 1897. He reviewed the papers of many who had advocated conservative operations—Siebenmann, Manasse, Bondy, Bárány, Heath, etc.—and found there was no precise indication for preserving the ossicles, though all were unanimous that the ossicular chain must be preserved to get the best hearing results.

In this country Heath, in 1904, urged conservative measures and drew attention to the importance of early operation to preserve hearing. He devised a special technique.

There were—and still are—opponents to the conservative operation. Some quoted cases in which the radical operation was required after the conservative had failed; others suggested that the good results of the conservative operation were due to the fact that no operation at all was required.

The conservative operation meant, for some, the preservation of all the ossicles, but others showed that excellent results were obtained by the removal of the incus as it was so often necrosed.

At one time the presence of cholesteatoma was looked on as a direct indication for a radical operation, now this is no longer so: Beyer, writing in 1926, stated that the radical operation is primarily called for in the presence of intracranial complications. It is not required in the presence of cholesteatoma as some small collections may be cleared out of the attic successfully. He advocated the attico-antral operation: this can be performed through a posterior incision or through the meatus.

Fraser also, in 1926, wrote: "Cholesteatoma does not necessarily mean a radical operation, though there is a slightly increased risk in performing the conservative operation when it is present." He considered the patient's safety first and kept the modified operation for cases with good hearing.

There are many different methods of performing the conservative mastoid operation: bone is removed from the posterior meatal wall far enough to allow meatal flaps; the outer attic wall is removed to drain the attic and observe the presence of cholesteatoma and the condition of the ossicles; some remove the "bridge," others leave just enough to support the membrane. If the attic wall is not removed the aditus is exposed and the condition of the incus investigated; if necrosed the incus is removed. The position of the perforation and the amount of hearing are determining factors in choice of operation.

In radical, modified radical, and conservative operations the treatment of the meatus has varied a great deal; it has been cut in various ingenious ways, thus making flaps to allow of free drainage into the meatus; now the tendency, in this country at least, is to leave it alone as much as possible. The tendency to narrowing of the meatus was countered by packing, tubes and skin grafts. In this country it was Sir Charles Ballance who perfected skin-grafting. To fill the bony cavity periosteal flaps have been advocated and skin periosteal flaps. Lately, fat grafts have been inserted, and flaps from the temporal muscle turned down into the cavity.

No survey would be complete without some reference to ossiculectomy, really a modified radical operation. A well-loved President of this Section, Hunter Tod, was most skilful in the performance of this operation; he made the very good point in its support—that it kept patients in hospital a few days only. Now, however, the operation finds but few, if any, supporters, and it is not mentioned in the most recent textbooks.

Lastly, let me sketch briefly some of the suggestions made to hasten the healing of the wound left by the operation for acute mastoiditis—the Schwartze operation or simple mastoid operation.

The wound is: (1) Left open and allowed to drain and granulate. (2) Allowed to fill with blood-clot. (3) Flushed by means of the Carrel-Dakin apparatus. (4) Closed by secondary suture. (5) Filled or wiped over with bismuth and iodoform paste and sutured. (6) Filled with ambrine, either at once or a day or two after operation. (7) Bone grafted after treatment with Carrel-Dakin fluid. (8) Treated by zinc ionization.

Though all these are ingenious and useful, the basis of success in this operation is through opening of all accessible cells and with this we shall I think all agree.

The real and final triumph of the mastoid operation will lie in the successful treatment of all cases of acute otitis media, if need be, by the Schwartze operation, thus eliminating chronic suppuration, and the necessity for radical operation wil come to be looked on as a sign of failure.

The Triumphs and Failures of the Mastoid Operation.

By L. Graham Brown, F.R.C.S.

In every mastoid operation the chief aim of the aural surgeon should be (1) to eradicate a pathological condition, which is, in varying degree, always a potential danger to life, (2) to obtain complete and relatively rapid healing of the wound, and at the same time (3) to preserve, as far as possible, the physiological function of the organ of hearing. According to what extent he is able to approximate to this ideal, will prove the measure of his success or failure.

The nature of the pathological condition present must necessarily greatly influence the results of operation. Experience has shown that it is far easier to remove an acute infective process from the middle ear and its adnexa than a chronic one. In the latter case, moreover, apart from the fact that the disease has become more firmly established, it has, owing to its chronicity, in most cases brought about some degree of irreparable damage to the delicate and essential structures of the sound-conducting mechanism, or even to the labyrinth itself. Hence, in the process of elimination of the chronic disease, it may often be found necessary to employ more drastic or radical methods of operation, methods which tend moreover to impair still further the hearing function. No operation, however, can be counted a success that destroys further or renders useless a previously useful organ. These facts alone will primarily determine that it is in the list of acute mastoidectomies that we must look to find our greater number of successes, whereas in the chronic our failures will be more prone to occur. Likewise they will determine what methods of intervention should be practised to deal successfully with the pathological condition present, and at the same time preserve as far as possible a useful auditory function.

To-day the simple mastoid operation—that based upon the earlier method of Schwartze—may, for all practical purposes, be considered the most efficient method of intervention in almost all cases of acute mastoiditis. Its essential principles consist in the classical post-auricular incision and exposure of the mastoid bone and a thorough removal of all diseased bony tissue, followed by a partial closure of the wound, after the establishment of free drainage from the cavity to the exterior. The tympanum and its contents, as also the tissues of the external auditory meatus, are all truly respected, since interference of any kind, excepting perhaps incision of the tympanic membrane for better drainage of the middle-ear, may, when resolution

has occurred, readily cause an impairment of hearing. Happily, in these circumstances, success can now be generally assured. The inflammatory process having had too little time to bring about irreparable damage to the delicate middle-ear structures, the simple mastoid operation, followed by careful after-treatment of the wound, is all that is necessary to ensure a real triumph for the surgeon. With a wound soundly healed, a scar scarcely visible, a tympanic membrane intact, and the power of hearing fully restored, the surgeon can proudly claim that he has caused no physical disability of that ear. How easily, however, can such a potential success be turned to failure, and what mistakes, not always directly attributable to the surgeon, can readily mar what might have otherwise proved a brilliant result! As we shall see later, the after-treatment of mastoid cavities plays almost as important a rôle towards success as the operation itself, and certainly must be counted as the greatest factor in the production of those post-operative conditions which we classify as failures. In due course reference will be made in this paper to a list of these failures, and an explanation attempted as to their various causes. I firmly believe, however, that many a mastoid operation for an acute condition has been spoilt by an unnecessary interference with the external auditory meatus through a mistaken idea that drainage thereby will be better effected. Apart from the possibility of the establishment of a meato-mastoid fistula, must we not assume that we are altering the formation of a structure that Nature has best adapted to the requirements of the hearing function? At one time the cutting of a meatal flap was a common practice in operating upon cases of acute mastoiditis, but of late years we have seen it fall more and more into disuse. Perhaps this is due to the fact that nowadays diagnosis is made more early and experience has taught the surgeon that drainage posteriorly by means of the simple mastoid operation will generally suffice. I believe, however, there are cases in which the employment of the meatal flap is still strongly indicated. I refer to those cases of mastoid disease, unhappily still too common, that have remained neglected since perhaps the third or fourth week of their origin. The infection, usually of a low-grade virulence, may give no other indication to an unsuspecting examiner than a copious aural discharge. When the aurist comes to operate, he finds a very extensive necrosis of the mastoid, usually cellular, and of the zygomatic cells, with œdema surrounding the meatus to such an extent as to cause almost complete stenosis. In such circumstances a small triangular flap of the soft terminal tissues of the posterior meatal wall will be found to aid greatly in the subsequent drainage of infection from the tympanum.

We come now to a consideration of the best operative procedure in dealing with cases of chronic suppuration of the middle ear and mastoid. Among these cases we naturally include those acute cases that supervene upon chronic conditions, since the operation planned usually seeks to eliminate not only the acute infection but the source from which it has sprung. A further problem now confronts us. We are dealing not only with a focus of disease, but also with a chronically impaired organ of hearing and possibly an impaired labyrinth. Again our aim is to eradicate disease completely, to secure a dry healed wound, and to conserve as much as possible the hearing function. What type of operation should be performed, and what are its indications? Surely the guide to our decision will rest upon the degree of useful hearing that is present and which we must endeavour to preserve.

Our latest conception of the physiology of hearing appears to lay more and more stress on the function of the membrana tympani and chain of ossicles as being one of auditory accommodation rather than that of sound conduction. On the free mobility of the two membranes of the windows of the labyrinth depends the normal functioning of the labyrinthine fluids in the conveyance of the sound vibrations to the receptor organs of the cochlea. Hence we may conclude that, provided these two membranes are kept mobile, the transmission of sound waves to the receptor

organ of hearing is by no means an impossibility, even though the remaining contents of the middle-ear have been partially or totally destroyed. This truth is borne out in the light of practical experience. Many a radical operation on the mastoid or middle-ear has left the ear with useful hearing. On the other hand, how many similar operations have resulted in a loss of useful hearing! One surmises that on the one hand, the mobility of these delicate membranes of the windows has been interfered with by some sclerosing process, whereas on the other, they have escaped by a happy accident or by design. In otosclerosis a similar cause for the loss of hearing can be traced to the disturbance of the normal action of the two membranous windows. If we accept this theory, we must admit that it is clearly an advantage when planning a radical mastoid operation, to consider in what way this region of the inner wall of the tympanum containing these windows can be conserved as nearly in their natural state as possible, and at the same time how to rid the cavity of disease. Obviously this will depend on the nature of the disease and the region of the tympanum mostly affected, and on what steps are necessary firstly to remove the diseased tissue and afterwards to provide for adequate drainage of the area. Fortunately in a great number of chronic suppurative conditions of the middle-ear and antrum—particularly in the simple chronic suppurations without granulations or cholesteatomatous formation, and in attic disease, which is as a rule confined to that region—a considerable portion of the tympanic membrane and mucosa of the tympanum can be preserved. The operative technique, therefore, takes into consideration these structures, and instead of the complete radical operation, some more conservative method will often meet the case, giving excellent results both as regards the removal of infection and the ultimate effect on hearing. Access to the attic can easily be obtained by removal of its outer wall and likewise the aditus can be opened in order to expose—and, if found diseased, to resect—the incus, without causing further destruction of the tympanic membrane and middle-ear structures, or even of the meatal walls. The postero-superior attachments of the drum membrane and of the terminal portion of the membranous meatus must necessarily be broken down on such occasions, but these succeed in finding attachment to surrounding structures again during the healing process.

When no useful hearing exists, the radical mastoid operation, with removal of the remaining contents of the middle-ear, can be undertaken, when obliteration of the Eustachian tube, if successfully performed, will well repay the surgeon's efforts towards obtaining a dry cavity. Again one advises, however, the smallest amount of interference with the meatal tissues consistent with that which will provide adequate drainage. In fact, a large meatal flap is now very rarely necessary and in my opinion should be reserved for those cases previously operated upon which have resulted in a stenosis with a persistent chronic discharge. This brings us to the question of the advantages, if any, of the skin, fat, and muscle grafts. Without a large meatal approach to the bony cavity skin grafts cannot be usefully employed, and hence should be reserved for that small minority, already referred to, wherein an attempt is being made to repair the results of a previous failure. In the same way fat and muscle grafts should only be found necessary in similar cases of failure with permanent antral fistulæ, deep depressions, or on those rare occasions in a primary operation when one fears that, owing to the large amount of bony removal, some such deformity may result. In such cases one would perhaps give preference to the live muscle graft turned down from the temporal muscle.

As the result of present-day teaching and from my own experience, I would say, therefore, that the tendency in mastoid surgery should be always to employ a method which, whilst approximating closely to the simple Schwartze operation, conserves as much as possible the meatal and middle-ear structures.

Before leaving this subject of the conservative surgical treatment of the chronic suppurations of the middle-ear, it may not perhaps be out of place to

refer briefly to the development of this idea. First conceived in Germany about 1900, as a reaction against the too frequent employment of the radical operation for all forms of chronic otorrhœa, and associated with the names of Körner, Stacke, and Siebenmann, this idea of conservative surgical treatment of chronic otorrhœa quickly spread over Europe and to America. Bondy and Lermoyez in France, Heath in England, Ballanger in America, and many other noted otologists, soon proposed and practised other modifications in the technique. Last year at the Otological Congress, held in Copenhagen, Professor Neumann in his report on the complete radical operation, brought histopathological proofs to show that the radical does not always put an end to suppuration, that this, even if it is temporarily cured, is subject to recurrences, and that the operation considerably alters the hearing function, if it does not lead to a complete deafness from secondary labyrinthine sclerosis. He showed the importance of conserving the smaller remnants of hearing, and concluded with a plea in favour of the more frequent employment of the conservative method. To-day our knowledge of this method is still further enriched by the work of Professor Maurice Soudille, whose conclusions drawn from his recently published paper can be very appropriately quoted as follows :—

(1) Every ear in which suppuration is prolonged for more than two months or recurring should be operated upon without delay if one wishes to save the hearing.

(2) On any occasion that a chronic auricular suppuration or one that has become acute, menaces in an urgent fashion the life of the patient, the complete radical is the operation of safety recommended.

(3) A chronic mastoiditis with fistula of the canal and integrity of the tympanum and Shrapnell's membrane should be treated by simple mastoidectomy.

(4) Regarding external attic suppuration or pure Shrapnell suppuration, experience has shown that the transmastoid atticotomy (i.e. opening into the antrum, aditus and attic via the mastoid) is the treatment of choice on condition that operation is undertaken before destruction of the ossicles.

(5) Finally, in chronic suppuration with perforation of the tympanic membrane—the most frequent condition—one must distinguish two kinds of perforation. (*a*) The small or medium-sized perforations with chain of ossicles intact : here a transmastoid attico-tympanotomy is indicated, with detachment and reposition of the tympanic membrane. This technique, which allows a wide examination of the tympanic cavity and an easy treatment of the lesions without altering a single essential organ of the apparatus of transmission, must be considered as an important acquisition : the healing of the suppuration, the closure of the tympanic perforation and the return to a hearing very near the normal, have become a possibility for many cases : only prolonged experimentation will be able to fix a definite value for this. (*b*) Large tympanic perforations with extensive caries or elimination of the incus and rupture of the chain of ossicles : the best procedure in these cases is the subtotal operation with removal of the incus and resection of the head of the malleus, conservation of the rest of the tympanum, curettage of the attic without curettage of the tympanum, no plastic on the canal. Post-operative treatment by the intermittent method of Carrel, for five to ten days, cauterization with chromic acid or zinc chloride in cases of granulations, then dressings with powders, ectogan, boric acid.

Among the post-operative conditions that may occur as the result of a mastoid operation the following must be definitely classed as failures :—

(1) Increased deafness with or without a persistent aural discharge, the latter often leading to its sequelæ : furunculosis, eczematous conditions and stenosis of the external auditory meatus. (2) Fistulæ (*a*) post-auricular ; (*b*) meato-mastoid, when a flap has been cut. (3) Unsightly scars, depressions, keloid. (4) Facial paralysis. (5) Intracranial complications.

The two latter are, fortunately, rare occurrences and must almost always be attributed to faulty technique on the part of the operator. An intracranial complication may arise owing to direct extension through an uncovered portion of the dura mater or sigmoid sinus. It is wise therefore, whenever the dura or sinus has been laid bare in operation—not in itself a very dangerous procedure—to render the site as completely aseptic as possible, enlarging the bony opening for that purpose if necessary, and to make sure that the post-auricular wound is left well open for purposes of inspection and drainage. The wound thus allowed to granulate may take longer to close, but the risks of intracranial involvement are thereby considerably diminished. Mention should also be made of the occasion when, during or after the removal of a large cholesteatomatous mass, infection, with its invariably fatal ending, takes place in a patient who, before the operation, had suffered perhaps little more than the ordinary discomforts of a chronic otorrhœa.

The remaining headings of this list constitute the more common classes of failures. As previously remarked they cannot always be laid at the door of the offending aurist. For instance, which of us has not seen a keloid or a permanent fistula arise to mar an otherwise perfectly planned and perfectly treated mastoid operation? Their ætiology, not yet fully understood, gives a loophole for our pride and we gain some satisfaction in the knowledge that their repair can very often be effected by some further form of plastic treatment. There is, however, another type of fistula for which we cannot claim the same indulgence. It may be either a simple post-auricular fistula, superficial from the mastoid cavity or even leading down to the antrum, or, when a meatal plastic has been cut for drainage purposes, the antral or mastoid cavity communicates with the external auditory meatus. Lined with an exuberant and infected granulation tissue, they may for many months prevent complete cicatrization and drying of the wound. They are the outcome of an incomplete operation or of a faulty technique in the after-care of mastoid wounds, or of both factors combined. It is essential in every mastoidectomy that a complete removal of all diseased tissue, with every possible focus of infection, should be undertaken, and this includes a thorough cleansing of the wound cavity and field of operation immediately afterwards. In this respect peroxide of hydrogen (10 vols.) has, in my hands, been found most satisfactory. The wound is now closed with adequate drainage and is in a condition to give the most happy results. Sometimes complete reparation immediately follows, the wound closing with scarcely any further attention within a week, the tympanic membrane healing, and a rapid and brilliant cure resulting to please both patient and operator. Not always, however, is one so fortunate. Re-infection of the wound cavity occurs, a fistula forms and we must now devote ourselves to a thorough routine of post-operative dressing of the infected cavity.

I do not wish to enter here into the controversy dealing with the respective merits of the various forms of post-operative treatment of mastoid cavities, whether it be by simple daily lavage with saline, the Carrel-Dakin method, continuous or intermittent, the employment of B.I.P. paste, ectogan, boric powder, alcohol, or various vaccines or even the application of paraffin or ambrine to the cavity. Each has its own enthusiastic advocate and results are published to prove the respective merits of all of them. We can content ourselves in laying down the broader principles that should govern our actions. Briefly, these comprise the maintenance of adequate drainage of an infected bone cavity without undue interference with the normal process of repair. To my mind, the simplest method of meeting these requirements is by daily irrigation of the wound cavity and meatus with some non-irritating fluid such as normal saline, boric lotion or Dakin's solution; and the reintroduction of a lightly-packed gauze ribbon drain. Fistulæ form owing to the fact that excessive granulations arise from irritation, pocket off pus in their folds, become themselves infected and thus interfere with free drainage and repair. The

occurrence of the meato-mastoid fistula makes us ask whether, apart from other considerations, the plastic on the meatus should ever be undertaken in the early acute mastoid operation, that is to say whether we should not content ourselves with the simple Schwartze method whenever possible. The answer to this, too, is clearly shown in the common practice of to-day, which tends to dispense more and more with this procedure. Likewise in the conservative and radical methods of operation we note the same tendency beginning to appear.

Depressions and unsightly scars after cicatrization are in themselves not very important results to justify the word "failure," as applied to mastoid surgery. Not always to be avoided, still so much the better if they can be, and this end can very often be obtained if sufficient care be taken in the planning of an incision, the proper excavation of the diseased bone, the suturing of the skin incision, and, moreover, in attending carefully afterwards to the healing of the wound. Perhaps, too, in their treatment a better future awaits the employment of fat and muscle grafts.

Moreover, in this list of failures, when a tympanic suppuration continues or recurs, let us not claim freedom from blame until we have assured ourselves that we have not neglected appropriate treatment of any exciting cause in the nose and naso-pharynx.

There remains that small percentage of cases in which, in spite of the most careful and appropriate form of treatment, failure may still result. Such cases must be considered as due to some idiosyncrasy on the part of the patient himself, who either possesses insufficient resistance to infection, or else has insufficient reparative power in his tissues. The latter is especially marked in certain types of sclerosed bone.

If, in the short time at my disposal, I have in this paper referred too briefly to the causes of failure in mastoid operations, and to the means at our disposal for their avoidance, I hope to be excused upon the grounds that I have wished rather to emphasize the points which may be raised later in the discussion, and to try to indicate the direction which our efforts must follow if, in the not too distant future, we are to speak with greater confidence and pride of the success that surely awaits us in the whole field of mastoid surgery.

The Radical Mastoid Operation.

By J. P. Stewart, M.D., F.R.C.S.Ed., and J. S. Fraser, M.B., F.R.C.S.Ed.

This paper gives an analysis of 401 radical mastoid operations performed by one of us (J. S. F.) in 395 cases at the Royal Infirmary, Edinburgh, during a period of ten years (1919-1928, inclusive). Modified radical mastoid operations and cases admitted with intracranial complications are not included.

An article published by one of us (J. S. F.) in the *British Medical Journal*, December 18, 1926, covers part of the ground dealt with in the present paper.

Sex.—212 males, 183 females. *Age.*—288 patients under 30 years. *Side.*—Right, 179; left, 210; bilateral, 6. *Cause.*—Only stated in 133 cases. Among these we may note: measles, 41; scarlet fever, 48; cold or influenza, 17; injuries of various kinds, 18.

In 305 cases the condition of the nose was noted, 243 were normal or showed only deviation of the septum; catarrhal or purulent rhinitis, 41; ozœna, 3; nasal polypi, 5; accessory sinus suppuration, 4.

Otoscopy.—Of the 401 ears operated upon, an aural polypus blocked the meatus in 116 and granulations in 59; sagging of the meatal roof was present in 30: a posterior marginal perforation was noted in 88, cholesteatoma and granulations in 32 cases, an attic perforation in 41, a large central perforation in 14, and an anterior perforation in only 7. In 14 cases more than one perforation was noted.

Hearing before Operation.—In 30, hearing could not be tested because patients were too young or too ill. Of the remaining 371, 10 were absolutely deaf on the operated side (four of these were deaf-mutes); deafness was severe (conversation voice at one foot or less) in 166; moderate (conversation voice at 1-4 feet) in 140; fair (conversation voice at more than 4 feet) in 54; and good (whisper at 6 feet) in only one case.

Vestibular Apparatus.—Of the 217 cases in which the cold caloric test could be carried out, the results were: A prompt reaction in 78; reaction delayed, i.e., between 40 and 120 seconds, in 105; in 24 the reaction took over two minutes, and in the remaining 10 there was no response. In 137 the rotation-test was carried out but the results need not be detailed.

Indications for Operation.—Aural polypi, 123: pain and mastoid tenderness, 43; cholesteatoma and granulations, 50; attic perforation, cholesteatoma and polypi, 33; sagging of meatal roof and subperiosteal abscess, 28; failure of conservative treatment (drops and syringing) 48; failure of zinc ionization, 36; failure of previous operation, 40.

Findings at Operation.—Superficial tissues normal in 326; a subperiosteal abscess was present in 21, a sinus in 15; in 39 cases more or less scar tissue was present. *Mastoid cortex.*—In 352, normal; in the remaining, 49 eroded by granulations or cholesteatoma, or former operation. *Mastoid process.*—Condition noted in 363 cases; dense in 356; pneumatic in only 3; in 4 cases (former operations) hollowed out by cholesteatoma. *Ossicles.*—Both healthy, 59; malleus healthy, incus absent or diseased, 115; malleus eroded, incus healthy, 4; both ossicles diseased, 116; both ossicles absent or not found, 84; not noted, 23. *Middle fossa and sinus.*—In only 6 cases was the dura of the middle fossa exposed by disease; in 8 cases the sinus was exposed by disease. In 39 cases, however, the sinus was far forward and therefore exposed by operation. *Mastoid antrum.*—Practically healthy in 48 cases; contained greenish-brown fluid with cholesterin crystals in 26; showed mucopus and swollen mucosa in 65; pus and granulations present in 129; cholesteatoma in a further 129. In the remaining 4 the antrum was extremely small or absent. *Tympanum.*—Contained polypoid mucosa or granulations in 261; a definite aural polypus was present in 64; cholesteatoma and granulations in 26; facial nerve exposed by disease in 3; in the remainder the condition was unfortunately not noted. *Skin graft.*—In 340 cases the operation cavity was lined by a skin graft.

Progress.—Uninterrupted recovery while in hospital, 314; unexplained fever for several days after operation, causing anxiety but with eventual recovery, 3; tonsillitis, 6; septic rash (surgical scarlet fever), 2; erysipelas, 6; stitch abscess or marked suppuration in posterior wound, 29; perichondritis (slight and severe), 7; giddiness and nystagmus to sound side (serous labyrinthitis), 7; purulent labyrinthitis, 6; temporary facial paresis present after operation, 8; severe hæmorrhage from cut edge of external meatus, 7; operation broken off on account of hæmorrhage, but completed later with success, 1; unsuccessful graft, 2.

In five cases death followed the radical mastoid operation; in all of these the cause was labyrinthitis followed by meningitis. In four of the five fatal cases a skin graft had been applied at the end of the radical operation.

The following table gives the length of stay in hospital, the results of operation, and the condition of hearing after operation, in the skin-grafted and non-grafted cases respectively.

Stay in Hospital.

	Grafted Cases. (Records of 323 cases out of 340)	Non-grafted Cases. (Records of 57 cases out of 61)
Less than 14 days	162 (50%)	17 (30%)
15—21 days	114 (35%)	27 (47%)
Over 21 days	47 (15%)	13 (23%)

Results of Operation.

	(Records of 216 cases out of 340)	(Records of 32 out of 61 cases)
Class 1.—(Cavity epithelialized) ...	74 (34%)	12 (37%)
Class 2.—(Outline of inner wall retained, but moist)	111 (51%)	16 (50%)
Class 3.—(Pus present with granulations)	21 (10%)	1 (3%)
Class 4.—(Much pus, polypi, etc.)	10 (5%)	3 (10%)

One patient had to be re-operated upon owing to membrane formation, pus and cholesteatoma.

Hearing after Operation.

	(Records of 216 cases out of 340)	(Records of 28 out of 32 cases)
Much improved	41 (19%)	6 (21%)
Improved	102 (47%)	12 (43%)
As before operation	28 (12%)	3 (10%)
Worse than before operation ...	26 (12%)	7 (25%)

(In two cases the patients were too young for any attempt at accurate information.
In 13 cases the hearing had not been tested before operation, and in two cases it was not tested after operation. Two patients could not repeat a shout close to the operated ear.)

Perhaps the first point that one deduces from these statistics is the great importance of preventing the acute ear from becoming chronic: "prevention is better than cure." Acute cases should never be lost sight of until a "dry ear" is attained. Certainly we must have the co-operation of the patient and his medical attendant to effect such a result, and here the sociological factor becomes apparent. We need only mention the relative number of cases of chronic suppurative otitis media encountered in private, as compared with that in hospital practice.

It will be seen that when the cause of the condition is stated, scarlet fever and measles are responsible for well over half of the cases. Here, again, we see the great value of an appointment of an otologist to the infectious diseases hospital. Continuing onwards to the findings under otoscopy, we note the comparative harmlessness of the anterior and central perforations. Taken together they roughly form 5% of the perforations noted in the cases which came to operation.

We draw attention to the fact that zinc ionic medication was carried out on 36 selected cases in the series without any appreciable difference to the course of the disease. Dr. Liston, who gave a summary of 46 cases of chronic middle-ear suppuration and 6 mastoid cavities which were treated by ionic medication in the Royal Infirmary, Edinburgh, records that 25·5% of them dried up. All that we are prepared to say is, that the method is worth a trial.

Aural polypi with symptoms were responsible for 123 cases coming to operation. In many of these cases the polypi had been removed on one or more occasions. Roughly 75% of polypi recur after removal. Thus, in the majority of cases of aural polypus, where there are no urgent symptoms, the simple removal is carried out more or less as a routine.

In only three cases was the mastoid process found to be pneumatic in type, thus supporting Cheatle's view that in chronic cases the mastoid process is of dense

or acellular bone. The density of the bone is due, according to Wittmaack, to infantile otitis media, and this density ultimately defines the course of the suppuration. Skiagrams have proved disappointing in giving any indication for operation. While in acute cases they are often of great value, in chronic cases they are all but useless, as they merely show the increased density of the bone, the position of the sinus, etc.

It is interesting to note that in 48 cases the mastoid antrum was found to be healthy, and in a further 26 cases there was very little disease found in that locality. This emphasizes the subjective or individual factor. If a patient with chronic middle-ear suppuration complains of earache we have no certain means of gauging the extent of his sensations; so pain, in the presence of a chronic suppurative lesion of the ear, has come to be regarded as a danger signal, and hence an indication for early operation. All of us must have encountered these cases in which pain is the indication for operation. In many of them we have been disappointed with the small amount of disease found at operation, but in the majority we have been profoundly thankful that we operated.

The incus is much more liable to disease than the malleus; this may probably be accounted for by its central and rather isolated position.

Now we come to the progress of the cases operated upon. Perichondritis is only encountered in 7 out of 401 cases. This tends rather to negative the idea that cartilage is of low resisting power and very prone to infection. It must be remembered that a Körner flap was cut in every case, that the incisions for it were made well into the cartilages of the auricle, and that a portion of cartilage was also dissected out from the base of the flap in order to diminish the possibility of contraction of the new external meatal orifice.

Undoubtedly the great danger of the radical mastoid operation lies in the occurrence of a labyrinthitis following the operation, the infection being apparently transmitted through the windows. This complication, together with its ensuing meningitis, was responsible for all the deaths in the series. When such a complication is diagnosed, the labyrinth should at once be drained.

The hearing of every patient is tested after operation without removal of the bandage, and with the noise-box in the good ear; further, any spontaneous nystagmus is looked for. It is not uncommon to have a minor degree of nystagmus to the opposite side from the operated ear on the morning following the operation, but the patient can still hear with the noise-box in the good ear. In all the fatal cases the labyrinth was opened and drained; the translabyrinthine drainage of West and Scott was also carried out.

The great majority of the cases (340) were skin grafted. This procedure has its champions and its opponents. The most outstanding difference between the grafted cases and the non-grafted cases from the statistical point of view is the length of stay in hospital, otherwise they correspond more or less. All must agree, however, on the absence of pain and bleeding at the first and subsequent dressings of the operation cavity in the grafted cases.

The skin graft also keeps the new meatus from contracting, and prevents the formation of granulations and the filling up of the operation cavity.

One of the two main causes of failure of the radical mastoid operation is patency of the Eustachian tube in spite of a thorough curettage at operation. The other chief cause is that many of the patients will not have their ears attended to after leaving hospital. We even go the length of providing them with ear syringes, but they soon tire of using them.

Discussion.—Mr. G. J. JENKINS said that in 1914 he was assisting a prominent surgeon at an ordinary Schwartze operation when the surgeon accidentally removed the incus by the posterior route. On testing the patient afterwards it was found that he had practically

normal hearing ; the tympanic membrane had healed perfectly. That incident had led him (the speaker) to think of its possible application in more chronic conditions.

The first of these cases he had was that of a German who was anxious to join the British Army in France. He was suffering from chronic otorrhœa. He (Mr. Jenkins) made the usual incision for the radical operation, and removed the incus from behind. The wound granulated in the ordinary way, as after a Schwartze operation, and the procedure was quite successful.

After the War he took up the subject again and the operation he did at first was as follows : The usual mastoid incision, only skin deep, was made, and the skin was then undercut.

He took a flap, using up the sterno-mastoid, which was pushed into the cavity after the incus had been removed. The object was to fill up the aditus and antrum completely, so that there would be primary union in the position indicated. Several surgeons saw that operation performed three or four years ago at a demonstration at King's College Hospital. Since then he had modified the technique.

The incision was the same as before, or a little further back. After undercutting the skin he cut a periosteum and muscle flap of triangular form with base to the pinna. The flap was well nourished by an artery that terminated at the apex of the flap. When the operation had been completed and the incus removed from behind, the apex of the flap was turned into the aditus and there was only a small space to be filled with blood clot. Immediate healing occurred, and it could drain in the position indicated. Looking into the meatus afterwards one could see no change of form, and a sound healing of the membrane occurred.

The most favourable kind of case was one in which there was a small perforation entirely within the membrane.

In all these cases the incus had been partially destroyed. In some cases he enlarged the opening in the tympanic membrane so as to improve the drainage. It was a valuable operation in cases in which the radical operation had been indicated, but in which, for certain reasons, as in children, it was important not to make any surgical disturbance.

Mr. E. WATSON-WILLIAMS said, he would confine his remarks to the radical operation because the only connection between the radical and the Schwartze was that they were performed on adjacent parts of the body. The Schwartze was for drainage, and was very useful in acute cases ; the radical was mainly for dealing with chronic suppurative conditions. In his experience, the majority of patients for whom one had to do the radical operation had not much hearing left, and one had the problem of a sinus leading down to carious bone. He had worked on the principle that in such a case one must not regard the ear as an ear, because ordinarily it was unlikely that one would be able to restore useful hearing when the patient had been deaf for years. The point was to get "exteriorization" of a hole in the bone. With that object he had tried to make the bone cavity as nearly saucer-shaped as possible, to cut down the facial ridge to the limit of safety, and to remove every scrap of mucous membrane. The plastic was made very open, almost the whole of the bowl of the concha being removed. A demilune of skin was removed entirely from behind the ear, so as to draw the pinna back, and bring the opening well over the centre of the cavity : finally, the edge of the periosteum was sutured to the cut skin of the bowl of the concha to anchor the ear in its new position. Dressing was easier, and this cavity had the great advantage, in hospital cases, that it was easily kept clean. The trouble with most of the patients who returned after a radical operation was the accumulation of epithelial débris and wax in the depths of the cavity, and the wider open the cavity the less frequently did those things occur, and the less was the trouble when they had to be dealt with.

Taking all classes of radical mastoid together, excepting only those patients who had died of intracranial complication present before operation (but including cases which came in with meningitis or labyrinthitis and recovered), he expected to get over 80% of dry ears, i.e., of ears which remained dry. He checked the cases by inspecting them or communicating about them two years afterwards. Of the remainder, half had only a small or intermittent discharge of moisture from the Eustachian tube.[1]

Dr. J. ALDINGTON GIBB said that he could not make up his mind from the discussion whether a post-meatal flap was desirable. He did a Schwartze in acute cases, but even in an

1 E. Watson-Williams, "Prognosis in Mastoid Disease," *Practitioner*, 1929, cxxii, 36.

acute case, when there was considerable destruction of bone, he made a flap; the best form of flap, he found, was Ballance's. Dressing was not easy in these cases, because of the tube behind the ear, but this could be modified. The patients in his wards had not suffered excessively when the wound was dressed. A drawback to Panse's flap was that if one did not look after the lower end of the flap and get good apposition, a bridge formed in the upper portion between the knot over the facial nerve, and had to be burned away. He did not think the muscle graft had advantages over the ordinary Schwartze; it would not obviate a radical mastoid operation. He only performed the radical operation when there was great destruction of the drum; he never touched the posterior wall of the middle ear if he could help it; during this operation therefore the stapes was always left in position. He had ceased trying to close the Eustachian tube, as the patient heard better if he did not do so. A patient who had been fairly deaf before the operation was able to take shorthand notes without difficulty; she could hear a loud whisper at a distance of five feet.

He had carried out Bondi's operation in a few cases, but he did not intend to do so again. At the end of a year or two the patient had a dry ear, perhaps too dry. Then he turned up again with middle-ear trouble. He had one with a large quantity of granulation in the middle-ear flapping over the Bondi's cavity.

Sir JAMES DUNDAS-GRANT said there must be some change in the cases seen at present compared with those Mr. Percy Jakins and he saw many years ago, as many more radical operations were performed in those days, he was sure, with very great advantage and saving of life.

With regard to the plastic operation, he had shown in March, 1924,[1] before the Section, a patient upon whom he operated about forty years ago, when the "plastic" was in its rudimentary stage. In those days, drainage was maintained in the posterior wound. He had also shown at the same meeting a girl for whom he had opened a temporo-sphenoidal abscess twenty years previously,[2] doing no plastic operation at all. The cavity was perfectly "papered" with a pink, smooth lining.

As to the skin graft, he once had to do the radical operation first on one ear and then on the other in the same patient. On the one side he skin-grafted. On the other side, owing to gouty phlebitis developing in one leg, the patient had to be kept very quiet in bed, so no grafting could be done. It was on the grafted ear that the patient depended for his hearing.

In his (the speaker's) early days he encountered many large cholesteatomatous cavities lined with a homogeneous matrix, and in many of those cases he had contented himself with making a wide opening and scraping out the contents, leaving the matrix behind. The results were very good. At a later meeting at Brighton, Hartmann praised this method of treating a cholesteatomous cavity. Such large cavities were seldom, if ever, seen nowadays, owing to better treatment in the early stages.

Only slight reference had been made to sub-periosteal abscess, which Gruber had described, but which Luc put forward as a novelty, at Boston, U.S.A. The swelling developed above the level of the auricle. The indication then was that the disease was not inside the mastoid, but on the surface; there should be a counter-opening made in the roof of the meatus, not through the mastoid.

It had been said that one indication for the radical mastoid operation was the presence of some intracranial complication. Some time ago he had the help of Mr. Ormerod in a case in which there was a lateral sinus thrombosis, which they were able to treat by ligation of the jugular vein, without clearing out the middle-ear at all; it was little more than was done for acute suppuration, and with complete retention of the membrane; that patient now heard well, and had a good membrane.

There was a tendency to decry the practice of ossiculectomy; he advised those who had not thought over the question to keep an open mind regarding it. He would show on a future occasion cases which would prove that there was still a place for the procedure.

He suggested a detail which had helped him to prevent discomfort from the changing of the dressing after the radical mastoid operation. Instead of plugging simply with gauze, he used one of the thin rubber finger-guards; this was perforated in numerous places and pushed into the cavity by means of Killian's narrow nasal speculum, through which the plugging was done. It could be left in for several days, and the perforations allowed the exudation to escape. When the first dressing had to be removed, there was no need of an anæsthetic, as there was no pain.

1 See *Proceedings*, 1924, xviii (Sect. Otol., 73).
2 *Ibid.*

Mr. SYDNEY SCOTT said Mr. Graham Brown's paper clearly expressed the principles of the mastoid operation, which he (the speaker) thought were likely to be followed. He agreed with the advice that a meatal flap should not be cut in acute cases. Mr. Heath, who had done so much good work in stirring the profession to the importance of mastoid operations, was the great advocate of the meatal flap, which he (the speaker) thought a disadvantage in acute cases.

Little had been said in this discussion about the methods of drainage. Some surgeons used gauze, some used rubber. The educated public believed the treatment after mastoid operation to be extremely painful, this impression he attributed to gauze packing. It was, no doubt, necessary to use gauze in cases of lateral sinus thrombosis, but in the ordinary uncomplicated Schwartze's operation he recommended suturing the upper part of the wound, and using several small rubber drainage tubes, without introducing any gauze into the wound. No anæsthetic was then necessary for after-dressings.

Mr. HAROLD KISCH said that with regard to pain after the use of muscle graft, he had been using a temporal muscle graft for some years, and had found that there was no pain after cutting the graft from the muscle; and, further, that the whole convalescence of the patient was painless. The only dressing used was a strip of gauze in the external auditory meatus, and the removal of that did not cause pain.

In reference to the question of obtaining sufficient muscle to fill a large cavity: there was no occasion to extend the mastoid incision, for, by pulling up the skin, one could obtain as much muscle from the temporal region as was needed to fill any cavity.

He had found that one point in the technique was not generally employed, namely, injections of novocain and adrenalin. In every case he infiltrated the skin, and then the deeper tissues, under the anæsthetic and before operating. The area of operation was found to have been rendered practically bloodless, a fact of great advantage in operating in these cases. The middle-ear and attic could be examined with ease.

Mr. E. BROUGHTON BARNES said he used only one tube for drainage—not gauze. He always quoted the remark in this connection: "If you want to use a drain you put in a drain; if you want a cork you put in a cork."

Recently, in cases in which the patients were not seriously ill, he removed the adenoids. He found that the mastoids healed more rapidly and the ear became dry sooner. He attended to the tonsils just before the patients left hospital.

With regard to exposure of the dura mater, he had been brought up to believe that this was a useful procedure if there was a fair-sized exposure and no thin edges were left. It enabled one to get a better-shaped saucer. He regarded the saucering-out of the cavity as a very important factor in quick healing and in the avoidance of fistula. He removed the tip as a rule.

Mr. F. W. WATKYN-THOMAS said that although he did not pack one ear in twenty in acute cases, in the last fortnight he had packed two cases of acute mastoid. He only did it for severe toxic mastoids, when he left the cavity widely open. His custom was to use two or three small tubes, as Mr. Scott had taught, though sometimes he employed rubber tissue. The latter, however, did not seem to work well, probably because it perished.

He was timid about removing adenoids at the time of the main operation, as to do so left an extra raw patch in the throat, over which pus could trickle down. He agreed, however, that adenoids should be attended to soon afterwards.

In mastoid operations for the more acute cases there was the question of removing the tip. A set amputation of the tip had, he thought, much to commend it, but he did not do it as a routine.

Mr. E. WATSON-WILLIAMS, in further comment, said his routine was to remove adenoids in every case at the end of the Schwartze operation: almost all child patients who needed the latter operation had large adenoids.[1] He had been quite satisfied with the results of that procedure.

Dr. J. S. FRASER said that he now saw fewer instances of chronic, foul-smelling otorrhœa than at the date (1906) when he was first appointed to deal with this work; he

[1] E. Watson-Williams, loc. cit.

hoped that as the time went on and acute cases were dealt with at the time they occurred, the radical mastoid operation would gradually fade into the background.

The number of radical mastoid operations which he had performed in the period 1906-1928 was 701. There was temporary facial paresis after operation in sixteen; serous labyrinthitis in thirteen, i.e., giddiness and spontaneous nystagmus to the sound side, though the patient could hear through the bandages with the noise-box going in the other ear. There was purulent labyrinthitis in ten cases. Seven patients died as a result of the operation, or rather from purulent labyrinthitis and meningitis following the operation. He had thought for a long time that the mortality of the radical mastoid operation was about one per cent.—he was not speaking of cases which came into hospital with brain abscess, sinus thrombosis, or meningitis; if those were included the figure would be brought up to 10 per cent. Of the 701 cases, 422 reported: 178 had a dry and satisfactory cavity, while in 244 the cavity was more or less unsatisfactory, i.e., either moisture was present (patent Eustachian tube) or granulation tissue could be seen. He was sorry to say that he could not get patients to keep their ears clean. If these cases were taken into hospital again for a fortnight and the ears were cleaned and washed out, a great many of them might be transferred from the unsatisfactory to the satisfactory category. Hearing was improved in 223: it was worse than before operation in only 64 cases.

Mr. GRAHAM BROWN (in reply) said that it rested with the younger school to further advance the field in this direction. Just as in the case of the accessory sinuses, he felt that there was room for more and more conservatism in regard to the mastoid. Much would be done towards this by getting the cases earlier. Indeed, the radical mastoid operation was now seldom necessary, and when it had to be performed it was in cases in which no useful hearing remained. The aim must be to conserve whatever hearing happened to be left to the patient. He thought one should in the future do a minute operation on the tympanic cavity; magnifying lenses were used, and there would probably be other appliances too.

With regard to drainage, though he still used a half-inch gauze ribbon, his method was not to employ it by plugging forcibly and so causing pain. Having practically closed the mastoid wound completely, he inserted an ordinary mastoid strip down to the antral cavity, and that was removed in forty-eight hours. After that, a sufficiently wide tunnel was formed for drainage. Afterwards there was gentle packing with half-inch ribbon gauze pushed down gently to the bottom of the sinus. Whether a tube or other form of drainage was used, the same applied.

With regard to exposure of the dura mater, it was not of great importance; he had had cases of it in which he had sewn up the wound completely, without fear of intracranial complications. One left the wound open only when one felt that the dura had been injured, in which case the extra time allowed for cicatrization was an added security.

Concerning skin-grafting and muscle flaps, he felt that the gain was not commensurate with the degree of destruction of the meatus. There were cases in which the cavity was dry after a radical operation, without interference with the meatus to any appreciable extent—except the terminal portion. In re-operating for mastoid trouble, however, when a large cavity was essential and a large meatal flap was necessary, skin-grafting proved very satisfactory.

A New Operation for Closure of a Post-Operative Mastoid Fistula.—DONALD WATSON, F.R.C.S.

This is a method of closing a post-operative post-auricular fistula. The diagrams, figs. 2, 3 and 4 (pp. 18, 19), are left-sided.

The first illustration shows a fistula and a wide curved skin incision about 1½ in. behind its edge and extending above and below away from the auricle, ensuring a good blood supply to the flap. This incision is made through the periosteum down to the bone.

The elevation of this flap is simple until dura mater is encountered. Here a cleavage is made in the fibrous tissue between the skin and subjacent bone or dura mater as the case may be. In these cases there is usually a good deal of dura exposed, and practically always the lateral sinus. The cleavage is made with the

FIG. 1.

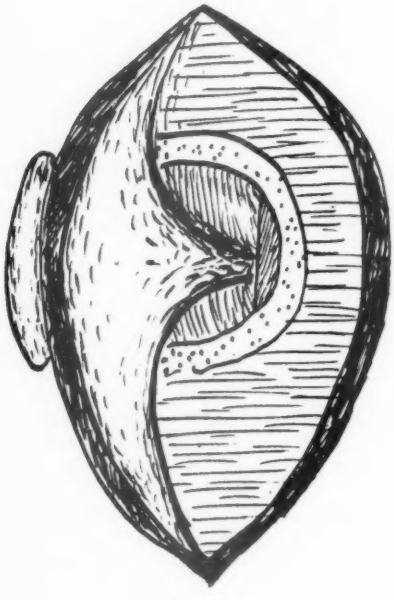


FIG. 2.

A new operation for closure of a post-operative mastoid fistula. The diagram (fig. 2) is left-sided.
(Mr. Donald Watson's Demonstration.)

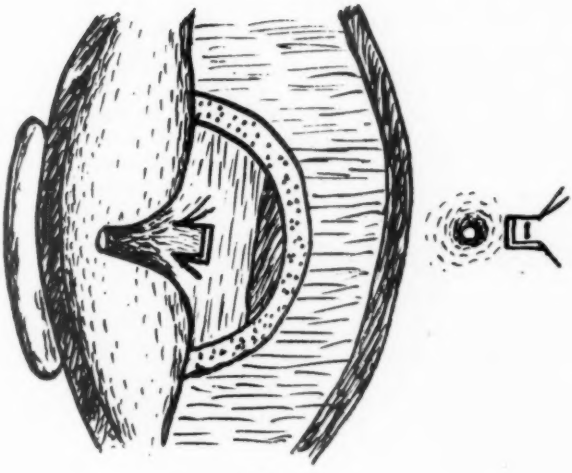


FIG. 3.

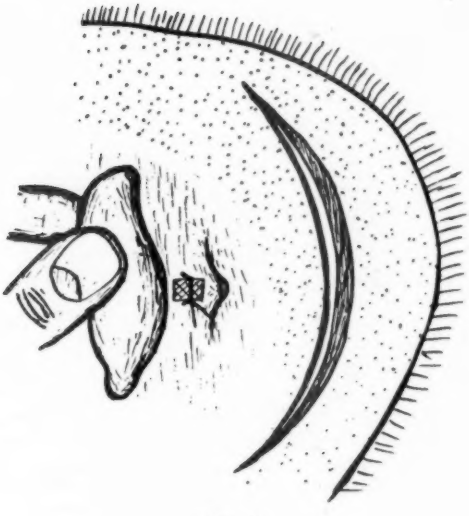
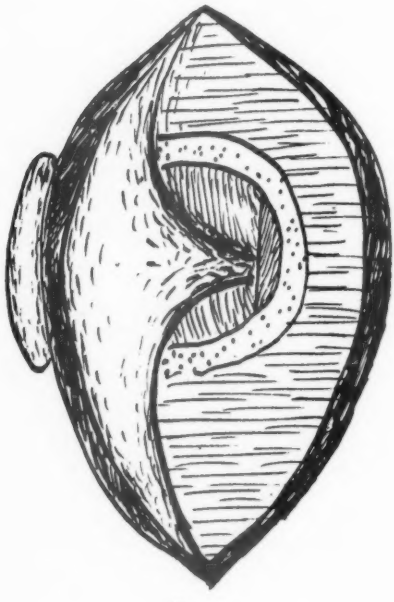


FIG. 4.

FIG. 1.



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A new operation for closure of a post-operative mastoid fistula. The diagram (fig. 2) is left-sided. (Mr. Donald Watson's Demonstration.)

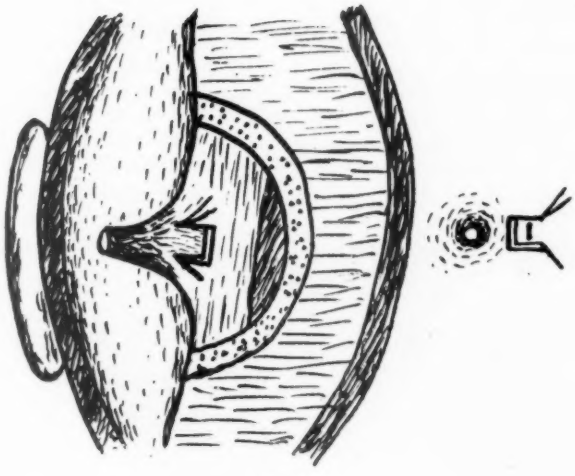

FIG. 3.

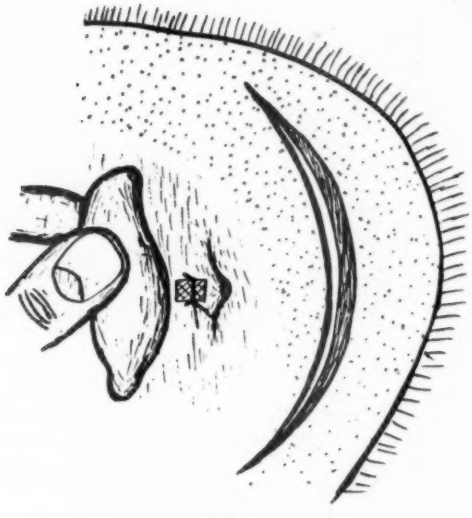

FIG. 4.

blunt edge of a dissector. I use an ordinary tonsil dissector after the manner of opening an envelope with a round lead pencil, occasionally giving a touch with a scalpel if the tissue is particularly tough. The fistula is isolated above, behind and below in this manner, as shown in fig. 2. It is now adherent to the posterior wall of the cartilaginous meatus anteriorly.

The next step is the cutting free of the fistula at its apex. The cut apex is grasped with forceps and dissected from the posterior meatal wall as high as is necessary. It now hangs free as a cone as shown in fig. 3.

A Körner flap is cut in the posterior meatal wall and stitched to the anterior wall or lip of the fistula by a mattress suture. The dissected fistula is now turned

FIG. 5.

inside out, as shown in the small diagram in fig. 3. By this manœuvre, the raw surfaces of the fistula are easily approximated and there is plenty of tissue to stitch and repair what was the fistula.

The mastoid cavity is then inspected and whether a radical (or modified radical) operation is done will depend on the circumstances of the case.

The cavity is packed with iodoform gauze wrung out in 1-1500 acriflavine and the original incision is stitched up without any strain on the stitches in the area of repair.

Fig. 4 shows the flap returned to position. The everted sinus appears as a cone and in front of it is a small pad of gauze to take the strain of the mattress suture of the Körner flap.

Fig. 5 shows the same case as fig. 1, eleven days after operation.

Three Cases of Conservative Mastoid Operation with Temporal Muscle Graft.—W. A. MILL, F.R.C.S.

(1) J. A., a boy, aged 6 years. For some years had left otorrhœa, which continued despite removal of tonsils and adenoids and treatment by drops and ionization.

Operation February 4, 1929, by Mr. Mollison.—Pus in cells, and granulations and pus in antrum; outer wall of attic and incus removed and muscle graft inserted. Result: hearing improved, no discharge.

(2) M. B., a girl, aged 10 years. For eight years had right otorrhœa, which continued despite local treatment and removal of tonsils and adenoids.

Operation May 27, 1929, by Mr. Mollison. Pus and granulations in antrum and in a few cells at the tip of the mastoid. Schwartze's operation with muscle graft. Result: hearing better. Discharge: none noticed; on examination—a trace.

(3) A. C., a girl aged 6 years. For some years had left otorrhœa, which continued after local treatment and removal of tonsils and adenoids.

Operation, March 18, 1929.—Pus and granulations in antrum. Many small cells containing clear fluid. Outer attic wall removed and the incus (the long process of which was missing) taken out. Muscle graft inserted. Result: hearing slightly better. Discharge used to pour out, now none runs out, but a trace appears on mopping.

I wish to thank Mr. Mollison for allowing me to show these cases as they have all been under his care at Guy's Hospital.

The procedure as adopted in two of them (1 and 3) is as follows: After a Schwartze operation has been performed, the outer wall of the aditus is removed to expose the incus. As a rule, bone has to be chipped off the postero-superior meatal wall (from the antral aspect). The incus is then removed because it is so frequently diseased (sometimes it is absent) and to allow of drainage from the region of the aditus and attic. The bridge is not removed.

The cavity is cleaned with hydrogen peroxide and then packed with gauze soaked in spirit. The muscle graft is cut in the way described by Mr. Kisch at the Summer Meeting, 1928.[1] By extending the incision about half an inch upward, and by retraction of the superficial tissues, as large a piece of temporal muscle as possible is cut and left attached anteriorly. The temporal fascia over this attachment is divided and sometimes a few fibres of the muscle—the aim being to get the cavity filled by muscle which lies easily without any tension.

The wound is then sewn up. If there has been much bleeding, a small piece of rubber-glove drain is left in the upper part of the incision for twenty-four or forty-eight hours. A tight bandage is then applied to stop the formation of a hæmatoma.

Dressing is reduced to a minimum and is practically painless; it consists of mopping-out the meatus and the installation of alcohol drops. The stay in hospital has been shortened and in several cases has been as short as ten days.

Results.—The muscle graft has been used in fifteen cases of chronic suppuration. In ten cases treated in the manner described (with removal of the incus) the ear is now dry in six (three of them being adults). Of these, three hear better than before, while in two there is no change and in one no report as to the hearing. In one of the four unsuccessful cases the ear was dry for about three weeks after the patient left hospital, but now discharges freely. In another a radical operation has

[1] See *Proceedings*, 1928, xxi, 1911 (Sect. Otol., 91).

since been performed. In the remaining two the discharge is very much less, while the hearing is better in one case and unchanged in the other.

The only case in which the incus was left is the second case exhibited. The hearing is very much better and, on mopping, only a trace of moisture is found.

Of four radical cavities which have been grafted (no plastic operation having been performed on the meatus) two are dry, the hearing being better in one of the cases and unchanged in the other. In the other two cases there is still a slight discharge. The hearing is worse in one of these and there is no report about the other.

Section of Surgery.

President—Sir HOLBURT WARING, C.B.E., M.S.

[*November* 6, 1929.]

DISCUSSION ON THE SURGERY OF THE SPINAL CORD

Mr. Hugh Cairns: I propose to limit myself almost entirely to a consideration of tumours of the spinal cord and adjacent structures, but I hope it will be possible to draw a few inferences that will be applicable to the surgery of the spinal cord in general.

Diagnosis.—The clinical course of the various types of tumour of the spinal cord is well known. Pain is usually the earliest symptom, either in the back or along the distribution of a spinal nerve. The pain may be neuralgic, burning, or tingling, or merely a tight feeling round the trunk or limbs. It is often aggravated by exercise, by coughing or sneezing, by straining at stool, by bending, or by lying down. Patients will sometimes volunteer the statement that they prefer to sleep sitting in a chair, because the pain is so much worse when they lie down. They will sometimes admit, when questioned, that their extremities are always cold, or that one foot is colder than the other, or that one foot is more sensitive to the heat of bath water than the other, but often pain is the only symptom for many months, and defies treatment by analgesic drugs or by other remedies for neuralgia, fibrositis, or rheumatism. In such cases successful diagnosis depends on the readiness of the clinician to perform thorough and frequent neurological examinations. Often he will be rewarded by finding one or more of the signs of an early and incomplete Brown-Séquard syndrome: an extensor plantar response and loss of postural sense in one leg, with disturbance of pain, heat or cold in the foot on the other side. Other symptoms that may occur before definite weakness sets in include stiffness of the legs, occasional collapse of one leg, numbness of one foot, "creepy" sensations about the ankles, slight urgency of micturition during excitement or during a particularly bad attack of pain. When symptoms have advanced so far as to interfere with walking, it is usually not difficult to make a correct diagnosis of tumour on careful clinical investigation alone. Important confirmatory evidence is obtained by means of lumbar puncture, when characteristic changes are observed in the composition of the cerebrospinal fluid, or blockage of the spinal subarachnoid space is demonstrated by means of Queckenstedt's test. Special stress should be laid on the importance of performing this test, which may give positive results at a time when the cerebrospinal fluid shows no change of colour and no appreciable increase of its content of protein. Its value as a post-operative test in estimating the results of treatment has hitherto been scarcely realized, and its use in the preliminary investigations is often neglected. It is still a common experience to be called to a case of spinal tumour which has undergone systematic investigation, and to find that, though the cerebrospinal fluid has been obtained and carefully examined, the simple expedient of attaching a piece of glass tubing to the lumbar puncture needle and measuring the pressure of the fluid has been neglected. Therefore, even at the risk of wearying you with what is already familiar, I propose to recapitulate the main features of the test.

If pressure is applied to both internal jugular veins in the neck during lumbar puncture in a normal individual, the cerebrospinal fluid at once drips much faster from the needle. When the pressure on the neck is released there is an immediate return to the slower rate of flow. This observation is based on the fact that cerebral venous pressure has a pronounced influence on intracranial pressure. Jugular compression causes the pressure in the cerebral veins to rise; there is a rise of intracranial pressure, the cranium cannot expand, and so cerebrospinal fluid, the most labile of all the contents of cranium, is displaced into the subarachnoid space of the spinal cord, and drips more rapidly from the

lumbar puncture needle.[1] If, however, the spinal subarachnoid space is blocked by a tumour at some point above the site of puncture, jugular compression produces no alteration in the rate of dripping (or pressure) of the escaping cerebrospinal fluid. This is the basis of the test of Queckenstedt, but in the test as now carried out, instead of trying to count the drops of spinal fluid, the observer attaches a straight piece of glass tubing to the lumbar puncture needle, and thus measures accurately the alterations of pressure that occur. In the normal individual there is an immediate rise of the column of cerebrospinal fluid when the jugular veins are compressed, and a correspondingly sharp fall when the compression is released. If no rise of fluid occurs when the jugular veins are compressed, it may be assumed that there is blockage of the spinal subarachnoid space somewhere between the foramen magnum and the point of the needle, unless, of course, there is thrombosis of both jugular veins or lateral sinuses.

Partial blockage of the spinal subarachnoid space by tumour sometimes occurs, and jugular compression is then followed by a partial and delayed rise of the cerebrospinal fluid in the manometer. There are certain precautions to be observed in interpreting the results,[2] but these are quite elementary, and the test is simple and accurate. It should be made as a routine in all cases in which tumour of the spinal cord is suspected. Jugular compression sometimes produces or increases root pains corresponding to the site of the tumour, and in this way the test may give additional information.

Diagnosis of the Level of the Lesion.—As surgeons we are particularly interested in the diagnosis of the position of a spinal tumour. There is nothing more unsatisfactory in spinal surgery than to overlook an easily removable tumour by exploring at the wrong level. In most cases repeated sensory tests will give an accurate indication of the upper level of the lesion, but they rarely give information about the extent of the lesion, and in a few cases they may actually mislead us into exploring at too low a level. In one of my cases the upper level of sensory loss was at the eleventh thoracic dermatomere, but a further examination after lumbar puncture, showed disturbance of sensation up to the fifth thoracic level. The tumour was subsequently found at operation to lie in the sixth thoracic segment of the cord. This case emphasizes the importance of repeating the neurological examination after lumbar puncture in the likelihood of obtaining further signs (Elsberg [1]). Sometimes the existing signs are grossly aggravated by lumbar puncture, as in a case described elsewhere [2], in which a mild weakness of the legs was converted into an almost complete paralysis. If this complication occurs after lumbar puncture, early operation is imperative.

The upper level of sensory loss should be tested not only by light touch, pin prick, heat and cold, but also by the tuning fork and by investigation of the patient's ability to appreciate numbers written on the skin (Zahlenschreibenerkentniss), for, as Foerster [3] has shown, the posterior columns are often affected in compression of the spinal cord more intensely and at an earlier time than the lateral columns.

Holmes [4] has pointed out that observation of the movements of the intercostal and abdominal muscles will sometimes reveal accurately the upper limit of the paralysis, and Feiling [5], in an admirable summary of the clinical features of compression of the spinal cord, has emphasized the importance of careful study of the tendon reflexes as a means of determining the level of a lesion of the cervical and lumbar enlargements.

All these methods should be employed, but it is important for those who are called upon to perform the operations to remember that the most careful clinical investi-

1 There may be other factors at work in producing this rise of cerebrospinal fluid pressure. The rise of pressure that occurs in the cerebral venous sinuses when the jugular veins are compressed probably stops the filtration of cerebrospinal fluid from the subarachnoid space into the venous sinuses. It is possible that there may even be a reversal of flow of the cerebrospinal fluid through the arachnoid granulations.

2 An excellent description of the technique of the Queckenstedt test is given in "Recent Advances in Neurology," by W. R. Brain and E. B. Strauss, London, 1928.

gations may be fallacious. It is rather significant that such an able and experienced neurologist as Professor Foerster [6] should almost invariably use the lipiodol test before he does a laminectomy in cases of spinal tumour. Most surgeons will now agree that the lipiodol test should be used not so much to diagnose the presence of a tumour, as to demonstrate the exact level of a tumour or other surgical lesion whose presence is already, on clinical grounds, certain or almost certain. It is especially indicated in those cases which show a shifting upper level of sensory loss. It tends to aggravate the pre-existing symptoms, and should therefore only be used when preparations for the operation have already been made. In that way the effect of unpleasant sequels, such as aggravation of a pre-existing paresis, is so much minimized as to become unimportant.

The lipiodol test is particularly valuable in those cases in which there is more than one tumour of the cord. In a case reported by Oljenick [7] the clinical diagnosis was tumour of the lower part of the cauda equina, but the lipiodol was obstructed at the level of the eleventh thoracic vertebra, and at operation two endotheliomas were removed through an extended incision, the one that had been diagnosed clinically and the other that had been revealed by lipiodol. In parenthesis, I am surprised that we do not more often see cases of multiple tumours of the spinal cord and its roots. For reasons which need not be mentioned now, we have made it a rule at the London Hospital, during the past eighteen months, always to remove the spinal cord in necropsies on cases of intracranial tumour. The number of cases in which unsuspected and frequently multiple tumours have been found in the cord is remarkable. Neoplasia of the central nervous system is probably much more commonly multiple than has hitherto been realized. When spinal tumours are multiple the difficulties of surgical treatment are not necessarily greatly increased, for the tumours are usually benign. The difficulty of clinical diagnosis is, however, tremendously increased, but the judicious use of lipiodol will clarify many diagnostic problems of this type.

Disturbances of Spinal Reflexes.—In considering the treatment of tumours of the spinal cord, it is necessary to inquire into the way in which tumours disturb the reflex activity and conduction of the cord. The study of spinal reflexes in man received great impetus from the paper of Walshe [8] which appeared in 1915. During the Great War the attention of neurologists was focused upon injuries of the spinal cord, and many excellent reports were published, culminating in the papers of Riddoch [9], and Head and Riddoch [10], which finally and conclusively showed that, contrary to previous belief, the reflex functions of the spinal cord below the point of complete section, return, or tend to return, after the phase of spinal shock has passed off. This discovery had considerable practical bearing on the management of all spinal cases. For the first time it became clear that the bladder of a patient who had suffered a complete transverse lesion of the cord could, under favourable conditions, empty itself completely by reflex action after spinal shock had passed off. If prolonged over-distension of the bladder and consequent destruction of many of its muscle fibres could be prevented during the first few days after the injury, reflex evacuation would soon begin.

Employing physiological methods at the bedside, Head and Riddoch were also able to show that the reflex functions of the cord, distal to the level of transection, were depressed, or their recovery was prevented, by the intervention of toxæmia, as from septic bed-sores, cystitis and ascending pyelonephritis. These complications were not only in themselves a potent cause of death in spinal lesions; in milder degree they delayed the recovery of spinal reflexes. Reflex micturition being imperfect, the bladder could not empty itself completely and this, in turn, tended to aggravate the urinary infection; for it is well known that a most potent cause of cystitis is the inability of the bladder to empty itself completely. Cystitis disappears and ascending pyelonephritis never occurs if the bladder is kept empty or

is emptied completely with each act of micturition [11]. There is no need to dwell on the numerous fatalities, due to ascending pyelonephritis, in cases of spinal injury seen so often in former days and still occasionally seen, not only in traumatic cases but also in tumours, through neglect of the simple rules that should govern the management of the bladder in all spinal diseases. Every paralysed bladder should be regarded as an infected bladder. Unless emptied regularly, not twice, but four times a day, it will sooner or later become severely affected.

A recent case of a complete transverse lesion at the fifth cervical segment provided a good example of the recovery of bladder function that may be obtained when the correct methods are employed. Within twenty-four hours of the injury the bladder was distended up almost to the umbilicus, but it was then emptied by catheter and thereafter was kept empty by a tied-in catheter, from which the cork was removed every four hours. Twenty-six days after the accident the patient expelled the catheter and at the same time passed 4 oz. of urine. Later in the day he passed urine automatically and immediately afterwards he was catheterized and the bladder was found to be empty. From that time onwards micturition was automatic. A catheter was passed every three days, but the amount of urine obtained never exceeded 4 oz. A gentle bladder wash was given at each catheterization. When last seen four months after the accident there was no return of movement or sensation, but the patient had remained free from serious cystitis.

Disturbances of Spinal Conduction.—Our main object in the treatment of spinal cases is to get the patient to walk again, and we are thus much more concerned with the recovery of the conduction of the cord than with recovery of its reflex function, even though the spinal reflexes are manifestly essential to the act of walking. So little is known about the disturbances of conduction of the spinal cord that I would hesitate to discuss them if there was not something of practical value to be gained. Anatomical division of fibres of the cord disturbs conduction, but this cause does not act in spinal tumours; as Purves-Stewart and Riddoch [12] point out, compression of the cord by tumour should be clearly distinguished from destruction of the cord by injury. From a study of the course of cases after removal of spinal tumours it is clear that conduction may be disturbed by a number of different causes.

In rare cases compression produces severe degeneration of the cord and no recovery of function occurs, even though the tumour has been removed. This happened in one of my own cases, in which an extradural sarcoma produced complete paraplegia within three days of the onset of the first symptom. Unfortunately, I did not see this patient until some time later, when he already had severe cystitis and numerous bed-sores. The tumour was partially removed at operation, and the remainder (as was subsequently proved) was completely destroyed by X-rays, but no recovery of function occurred. The patient died from bronchopneumonia and septicæmia three months and a half after operation. At necropsy, though the tumour was found to have been completely destroyed, the cord showed massive ascending and descending degeneration from the site of compression, which was itself severely degenerated. The treatment in this case had not been given soon enough. Sudden rapid compression of this type is fortunately rare. In the necropsy material of their series of spinal tumours, Purves-Stewart and Riddoch [12] found remarkably little degeneration in the cords of patients who had shown severe paresis in life. This is what would be expected from the clinical experience that the cord usually recovers part or the whole of its conduction after removal of a tumour. The damage to the conducting fibres is not so severe as to be followed by degeneration. It is a reversible process.

The nature of this reversible process has not yet been conclusively demonstrated. From a study of the morbid anatomy, Purves-Stewart and Riddoch consider that it is a vascular disturbance of the cord, a relative anoxæmia, at and below the site of compression. As far as I am aware, no experimental work has been done on

compression of the cord, but the evidence of experimental compression of peripheral nerves suggests that the process may be a purely mechanical one. Gasser and Erlanger [13] have shown that when a peripheral nerve is compressed, the larger fibres—the fibres conveying motor impulses and sensations of contact—are blocked before the smaller fibres, which convey sensations of cold and warmth. They have not studied the mechanism of this disturbance of conduction, but they assume that it is due to collapse of the fibres, the larger fibres collapsing more easily than the smaller, as any series of tubes of equal strength might be expected to do when subjected to compression.

The problem will probably be solved in the physiological laboratory, but it must first be clearly stated, by collection of careful clinical observations of the way in which recovery of conduction may occur after removal of spinal tumours. The examples that I have been able to observe indicate that the process varies remarkably in its course. Sometimes recovery of function in the paralysed limbs begins to be evident within a few hours of the removal of the tumour, and rapidly and in dramatic fashion becomes complete; at other times it is delayed a few weeks, yet finally becomes complete; in other cases it may only begin months or even a year or more after removal of the tumour, and even then may be slowly progressive.

Recovery of function is most easily observed and measured quantitatively in the upper limbs. Some interesting observations were made on a case of cervical tumour that I saw while at Dr. Harvey Cushing's clinic. The tumour was a meningioma lying on the left side of the spinal cord between the fourth and fifth cervical roots. Before operation the patient had complete paralysis of the left arm and leg, and only feeble movements of the right arm and leg.

TABLE I.—RETURN OF POWER (GRIP) IN THE HANDS, AS SHOWN BY ERGOMETER (IN KILOGRAMMES).

	Right	Left
April 22 (before operation	0—1	0
,, 30 (operation)	0	0
May 1 (24 hours after operation)	7—8	0
,, 2	8	0
,, 3	9—12	1
,, 4	11	1
,, 5	20	1
,, 7	20	2
,, 9	25	2
,, 10	25	2—3
,, 12	25	3—4
,, 16	—	5
,, 25	22	5
June 2	25	9
,, 29 (discharge)	28	22

Immediately after removal of the tumour, under local anæsthesia, the patient was conscious of increased power in his right side and was able, without assistance, to scratch the end of his nose with his right hand, a thing that he had not been able to do for some time before operation. The power of the hand grip was measured frequently with the ergometer by my colleague, Dr. John Fulton. Table I shows how recovery of voluntary power began in the right hand as soon as the tumour had been removed, while in the left hand the recovery was delayed. No appreciable improvement began there until more than three weeks after operation, but then it progressed so well that two months after operation the left-hand grip was almost as strong as the right.

My own views on the recovery of patients after the removal of spinal tumours were revolutionized last summer when I had the privilege of visiting the clinic of Professor Foerster at Breslau, and of observing his remarkable work on the surgery of the spinal cord. Professor Foerster has operated on a number of apparently hopeless cases of spinal compression and has kept them in hospital for long periods—even for years—after removal of the tumour. He has kindly supplied me with the

notes of some of his cases, which show how important the quality of patience may become in the treatment of long-standing compression. Thus, one case of paraplegia in flexion had been treated for six years as a case of disseminated sclerosis. Professor Foerster removed a tumour attached to the first thoracic nerve root, and during the next fifteen months he straightened the legs by movements under a series of anæsthetics. Eventually the patient walked well. In another case, that of a woman who had been confined to bed for four years with complete paraplegia in extension, the tumour was embedded in the side of the cord and, after its removal, the cord at the site of compression was so much thinned as to resemble a piece of paper. Recovery did not begin until over a year after the tumour had been removed, and then progressed slowly and gradually until six years after operation, when the patient walked again.

These observations are so encouraging as to make one feel that no case of spinal tumour is hopeless. In cases of long-standing compression, patience in the after-treatment is essential and every effort should be made to procure adequate institutional and orthopædic treatment during the period of recovery.

Blockage of the Subarachnoid Space.—Although we know very little about the disturbance of conduction in spinal tumours, a study of the condition of spinal arachnoiditis, or meningitis serosa circumscripta, provides good evidence that blockage of the spinal subarachnoid space plays some part. The clinical picture of spinal arachnoiditis is almost identical with that of a slowly developing spinal tumour, but the spinal fluid usually shows no increase of protein (Stookey [14]), and at operation, instead of a tumour, there are seen numerous adhesions of variable extent and dimension between the arachnoid and pia, and the arachnoid and dura. If the adhesions can be thoroughly broken up at operation the patient recovers, in spite of the fact that in the few cases which have come to necropsy, gliosis and scattered degeneration of the cord have been found (Horsley [15]). The pathology What is important to our present thesis is the fact that before operation, a partial or of the condition is by no means clear, and I do not propose to discuss it here. complete blockage of the subarachnoid space can be demonstrated by means of Queckenstedt's test, and that, after division of the adhesions, recovery of function in the paralysed extremities ensues. These observations strongly suggest that blockage of the subarachnoid space by adhesions interferes in some way with the conductivity of the cord. In many cases there is no evidence at operation that this interference with conductivity is due to the collection of fluid under tension in the loculi of the subarachnoid space. Stookey [14] has advanced the view that the adhesions fix the cord and interfere with its normal respiratory movements and with the very slight movements that it goes through during movements of the trunk. He believes that this constant interference with the normal movements of the cord, continued through a long period of time, can eventually produce disturbances of spinal conduction. This view of Stookey's receives support from the well-established experimental observation that tension on a peripheral nerve will block conduction. Mr. Bryan Matthews informs me that tension of about 0·25 gm. stops conduction of the small nerves of a frog. Conductivity is restored when the tension is removed.

The fact that adhesions and blockage of the subarachnoid space interfere in some way with spinal conduction is of great importance in the treatment of tumours. My surgical experience with spinal tumours has been that satisfactory results from operation are only obtained if this obstruction to the circulation of cerebrospinal fluid is removed. The re-establishment of a free circulation of the fluid can be seen in most cases at operation after the tumour has been removed, but a much better way of investigating this is to do Queckensted's test after operation. I would suggest that this test should be done as a routine after all operations on spinal tumours, and particularly in those cases in which recovery is not proceeding at the expected rate. The subarachnoid space may still be blocked after removal of the tumour, either

because the removal was not actually complete, or because there are some adhesions at the site of operation, or because there is another tumour elsewhere in the spinal canal.

X-rays and Radium.—A case described in an earlier part of this paper provides convincing proof that X-rays can destroy an extradural sarcoma. X-ray treatment is rarely effective in other types of primary tumour. The destructive action of X-rays is probably in the first instance accompanied by swelling of the tumour cells, and it should therefore not be employed except after a liberal decompression of the cord and at first only in small doses. Elsberg [16] has reported some adverse results which may have been due to swelling of the tumour through the use of large doses.

Dr. John Fulton and myself have recently been investigating the effect of radium emanation on the cord, partly to find out whether radium could be used with safety in the treatment of malignant tumours near the spine. The results of our experiments will be reported in detail at a later date, but already it is evident that the effect of the emanations on the cord depends on the dosage employed, and that it can be predicted with remarkable accuracy. The experiments also suggest that radium emanations in small doses can be used with safety at a distance of 1 cm. from the cord. Large doses applied outside the dura mater produce complete and permanent destruction of the cord, and great care must therefore be exercised when it is proposed to place radium near the spinal cord.

Injury to the Spinal Cord at Operation.—It has long been known that the cord is a most delicate and vulnerable structure, to be respected at operation, even more than the brain. Knowledge on this point has grown up through the experience of operations in which the cord was too freely manipulated, so that its conductivity was afterwards lowered. It is now clear that with proper technique and care, the patient whose spinal cord is exposed at operation should not show any appreciable depression of spinal function as a result of the operation, unless the cord is deliberately injured in order to remove a tumour completely. Experience has also shown that a clean operative incision into the cord in certain situations is not attended by incapacitating or unexpected loss of conductivity. Thus, division of the antero-lateral columns of the cord for relief of otherwise intractable pain is not followed by any disturbance of conduction apart from loss of pain, heat and cold. Allen [17] has shown that a clean longitudinal incision of the posterior columns produces no demonstrable post-operative defect in dogs, and his experimental results are borne out by operations on certain intramedullary tumours in man. In one of my cases, that of a woman aged 29 years, who had suffered for about eighteen months from a gradually advancing quadriplegia and ataxia that eventually confined her to bed, it was possible to incise the posterior columns to the left of the mid-line from the third cervical to the third dorsal segment and to remove completely from the anterior aspect of the cord a circumscribed ependymal glioma weighing 14 gm., without producing anything but temporary depression of spinal function. For three days after operation there was further weakness and incoördination of movement, especially in the left arm and leg. After that day function was quite as good as it had been before operation, and it continued steadily to improve so that within three months the patient was able to walk and to use her hands more freely than she had done for months before operation, though postural sense in the legs was still completely lost. It is interesting to note also that there was no retention of urine.

It is thus evident that the type of damage at operation which produces severe disturbance of conduction is not of the nature of a clean incision. It is due to the pressure of bone forceps or gauze swabs during exposure of the cord, or to undue traction on the cord during exposure or removal of a tumour. And, if we may apply the observations made on the conductivity of peripheral nerves, it seems certain that traction on the cord, as during rotation, will interfere with conduction, even

though it is not so severe as to produce hæmorrhage or anatomical division of nerve fibres.

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Mr. Geoffrey Jefferson : The surgery of tumours of the spinal cord is in general the surgery of the relief of compression and as such is on as firm a basis as anything in medicine. The actual steps of the operation are no longer open to any important discussion, having become stereotyped, though individual cases may present certain problems. There are, however, three points on which we are still gathering information, and to these I shall briefly refer.

First, the place which Queckenstedt's test occupies, or should occupy. In my hands it has proved to be of very great value, but, as with all accessory tests, the results can only properly be estimated in correlation to the carefully elicited and recorded history of the case. The clinical history will always remain the most important guide in diagnosis here as in other regions. With increasing experience of jugular compression, the observer comes to recognize minor degrees of cerebrospinal stasis and block, short of complete obstruction, and a manometer is, of course, a necessity. The counting of drops issuing from the lumbar puncture needle will not suffice. This test is surely one of the most beautiful physiological demonstrations which one can give, but it must be linked up with the chemistry of the cerebrospinal fluid before we have the complete picture. This is an important point. The test can give wrong information if the apparatus is not perfect. In one case of my own it appeared that incomplete block was present, but operation failed to disclose tumour. The test was repeated three weeks later with a larger-bored needle and no block was found. It may be of course that the patient had an undiscovered intramedullary tumour, and that the laminectomy had given more room and abolished the block. But he may have had no tumour at all.

On a previous occasion[1] I exhibited a number of graphs showing different degrees of subarachnoid obstruction, and I was criticized for having laid too much stress on this side of the question and not enough on pain. Up to a point this was a just criticism; it is true that the typical neurofibroma of the posterior root is characterized by root pain which exists for some time as the only symptom. Later on, signs of cord compression make their appearance. It has been truly said that any unexplained local pain of radicular type may be due to a root tumour. But we must remember that this is the characteristic of one kind of spinal tumour only, albeit the most satisfactory to deal with and the commonest. We shall do wrong, however, if we say that all cases of spinal tumour exhibit pain as an outstanding characteristic. If the growths arise from a nerve root, or from the dura in the neighbourhood of the root, pain must be present. But if they arise

1 See *Brit. Med. Journ.*, 1928 (ii), 200.

elsewhere, pain by no means necessarily occurs. And there comes the danger, from too great insistence on pain as a necessary companion of spinal tumour, that cases may be missed because pain has not been a leading or outstanding complaint. I do not wish to be recorded as saying that spinal tumours do not produce pain, but I do ask that we recognize the meaning of pain in relation to the intradural structures, and that tumours can occur under the clinical guise of slowly increasing spinal compression, without root pain. In one case of my own I was able to enucleate an extradural tumour at the level of the first thoracic vertebra. This was in a boy, aged 12 years, and the only pain had been a vague aching in the thighs which had been regarded as growing pains. In another case a young man in the early twenties had a giant tumour of the cauda equina filling the dural sac from the level of the first lumbar to the first sacral vertebra. This tumour entangled all the nerve roots of the cauda equina, but the only pain had been some of slight degree in the left groin. The boy did not make a point of this pain and would never afterwards admit that it had been of any severity or moment. He had wasting of buttocks and thighs, with foot-drop and bladder disturbance, and had recently been discharged from the Army as a case of hysteria.

The next point is the use of lipiodol. We are all aware that it causes some reaction and its chemistry gives us reason to suspect it of power of damage, but the cases in the literature which are supposed to illustrate the damage that it has done do not bear close scrutiny. It is interesting to note that Tracey Putnam's work on the halogen-oil compounds seemed to show that air caused more reaction than most of the compounds though, to be sure, the air remains a very brief time as compared with the oil. I have myself regretted not using it on occasion. I feel that if the chemical examination and Queckenstedt's tests on the cerebrospinal fluid confirm the clinical history and physical signs and point to a diagnosis of spinal tumour, lipiodol may be used with advantage. If one is not sure whether a tumour is present or not, it is better to wait for a few months and re-examine, then to use lipiodol at once. For if the tumour is small, the lipiodol might go by and prejudice the correct diagnosis of that case for ever.

The third point is a purely surgical one: the correct handling of the intramedullary tumour, a point on which much further experience is needed and which might be made a subject for special discussion at some time in the future. Mr. Cairns is to be congratulated on his case.

The last point is the vomiting that is said to follow disturbance of mid-thoracic tumours. I have heard it said that this is due to the upsetting of the vegetative centres in the thoracic cord, and this may be true. But there is another possible cause, namely, uræmia due to renal infection. A young woman was referred to me by my colleague, Dr. Sturrock. She had lost the power in her legs within a few days, but progressively, not suddenly. I found at operation a diffuse sarcoma involving the laminæ in the mid-thoracic region and the muscles of the back. This sarcoma was completely cured, so it seemed, by external prolonged application of radium, but power did not return to the legs. After a few months the patient began to vomit and her abdomen became distended to such a degree that intestinal obstruction seemed a possibility. Her blood-urea, however, was found to be enormously increased and she died from uræmia. Exactly the same thing happened in another case not operated upon, that of a young woman with a secondary deposit of carcinoma in the spine presenting the same signs with the identical blood-urea findings. It is probably therefore of some importance in cases of spinal tumour to examine the blood-urea before operation.

Mr. George F. Stebbing: I had hoped to hear something of the operation of antero-lateral section. This is an operation which I have used many times for the relief of pain in cases of malignant disease of the pelvis, tabes dorsalis and

endarteritis obliterans. In every case there has been a great measure of relief, and in some cases the relief has been complete. I have found, however, that in the tabetic cases the pain has always recurred after an interval, the shortest interval being just under a year, and the longest four years, after the operation. In the cases of malignant diseases the relief has lasted as long as the patients' lives. There have been no evil results of the operation except in two cases in which the patients died, one from pneumonia two days after the operation, and the other on the sixth day from paralytic ileus. The patients have been able to get up and walk after the operation, although some of them had temporary paresis, which in no case lasted as long as three weeks. This paresis was, I think, due to handling the cord. None of the patients were incontinent after the operation except one who had been incontinent before it.

Mr. J. Paterson Ross said he had had two cases from which he had been able to learn a lesson which had not been mentioned during the discussion. The first patient was a woman, aged 45, with a history of gradually increasing paraplegia. An extradural neurofibroma was easily—and apparently completely—removed from one of the nerve-roots, and the dura mater was not opened. No recovery followed the operation, and the patient had gradually become worse.

The second patient was a man, aged 32, with a similar history and a similar extradural tumour. On this occasion, however, remembering the previous experience, it was decided that the dura should be opened, though the tumour appeared to be completely extradural. On opening the dura he had found a larger portion of the " dumb-bell " tumour compressing the cord. The connection between the intradural and extradural portions was extremely slender. Recovery from paraplegia was rapid.

It would be wise always to open the dura to look for an intradural portion of a tumour which might appear at first to be completely extradural.

Mr. Hugh Cairns (in reply) said he agreed with Mr. Jefferson that pain was sometimes absent in spinal tumours. He had seen cases of diffuse gliomatosis of the spinal subarachnoid space, secondary to medulloblastoma of the vermis, in which pain had been entirely absent. Of great interest were the cases in which pain was referred to the distal parts: in one of his own cases of intramedullary cervical tumour, the first symptom of any severity had been pain in the vagina. In the common tumour of a nerve-root, pain in the legs was sometimes a prominent symptom, even when the tumour was in the upper thoracic levels.

He had no doubt that Elsberg's extrusion method of dealing with intramedullary gliomata was the only way in which some of these tumours could be treated, but, in his experience, gliomata of the cord varied in their malignancy just as did gliomata of the brain. Some were quite circumscribed and of slow growth, and for these Elsberg's method was not indicated, because the capsule of the tumour was connected with the surrounding cord by numerous fine adhesions. Tumours of this type could be completely removed by gentle dissection.

Dr. Stebbing's experiences with cordotomy were of great interest. Professor Foerster had also had cases in which the pain recurred after complete division of the antero-lateral tracts. If these observations were confirmed it would seem that pain sensations might, under certain circumstances, be carried by the posterior columns.

In his experience, and in the more extensive experience of Stookey, the protein content of the spinal fluid was not raised in meningitis serosa circumscripta. He agreed with Mr. Paterson Ross that the dura should always be opened after removal of an extradural tumour, because of the occasional presence of an intradural extension of the tumour, or of adhesions which would interfere with the flow of cerebrospinal fluid.